"CLINICAL PROFILE AND SEVERITY OF STROKE IN DIABETIC AND NON-DIABETIC PATIENTS"

Dissertation submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

in partial fulfillment for the Degree of

DOCTOR OF MEDICINE - BRANCH I GENERAL MEDICINE

APRIL 2016

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI – 11, TAMIL NADU

CERTIFICATE

This is to certify that the Dissertation entitled **"CLINICAL PROFILE AND SEVERITY OF STROKE IN DIABETIC AND NON-DIABETIC PATIENTS"** submitted by **Dr. VENKATESH .R** to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment for the award of M.D.Degree(GENERAL MEDICINE) is a bonafide work carried out by him under my guidance and supervision during the course of study 2013-2016. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

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DECLARATION

I solemnly declare that the dissertation titled "**CLINICAL PROFILE AND SEVERITY OF STROKE IN DIABETIC AND NON-DIABETIC PATIENTS**" is prepared by me.

The dissertation is submitted to The Tamilnadu Dr,M.G.R.Medical university towards the partial fulfilment of requirements for the award of M.D.Degree (Branch I) in General Medicine . I also solemnly declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any university, found either in India or abroad.

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PROTOCOL TITLE: CLINICAL PROFILE AND SEVERITY OF STROKE IN DIABETIC AND NON-DIABETIC PATIENTS.

NAME OF PRINCIPAL INVESTIGATOR: Dr. R.Venkatesh, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: Post Graduate in MD General Medicine
DEPARTMENT & INSTITUTION: Department of General Medicine, Tirunelveli Medical College

Dear Dr. R.Venkatesh, *The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 14.05.14.*

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
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12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
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   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
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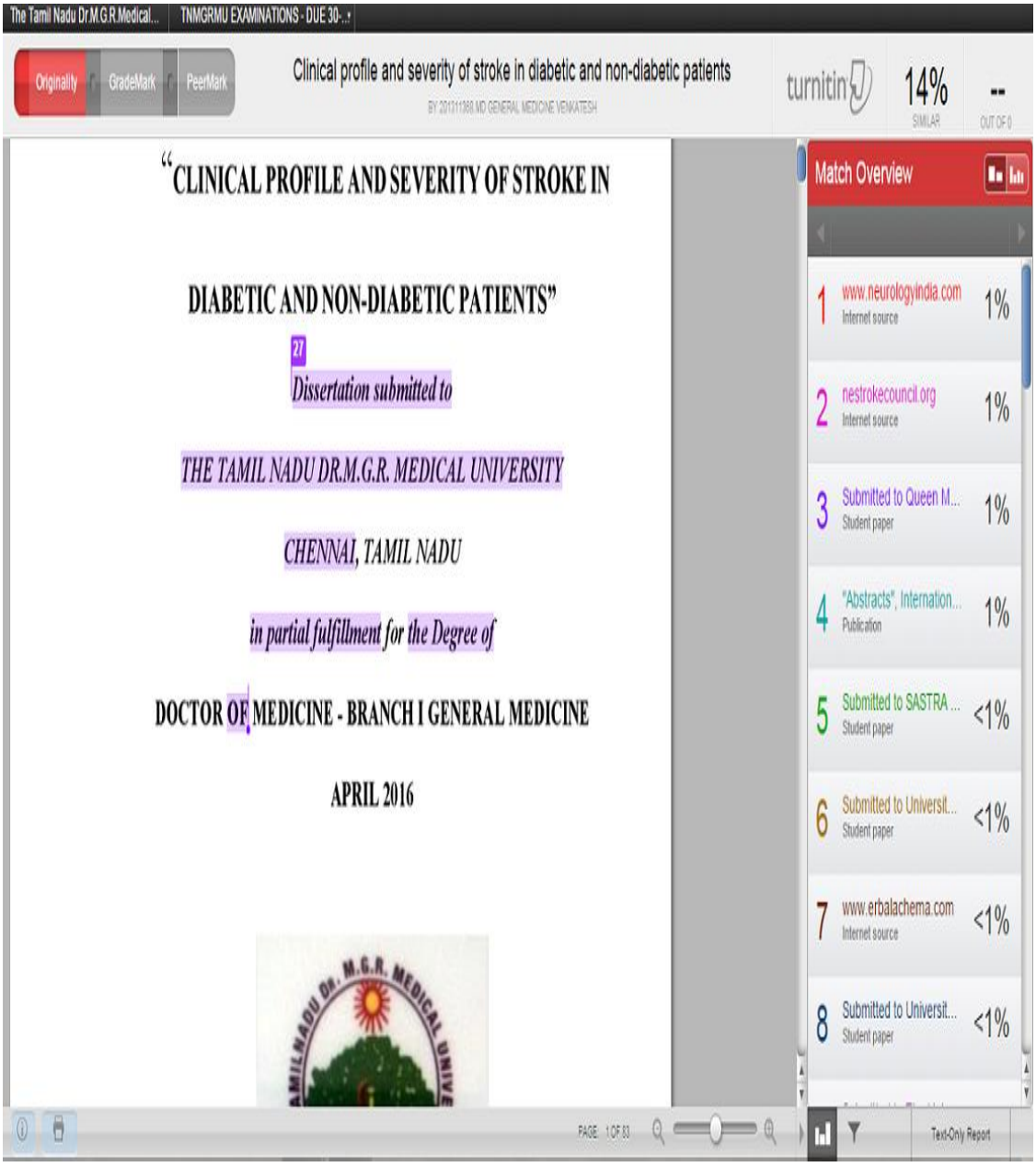
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CONTENTS

ABBREVIATIONS:

ACA – ANTERIOR CEREBRAL ARTERY

MCA- MIDDLE CEREBRAL ARTERY

PCA- POSTERIOR CEREBRAL ARTERY

NIHSS-NATIONAL INSTITUTE OF HEALTH STROKE SCALE

DM- DIABETES MELLITUS

IGT- IMPAIRED GLUCOSE TOLERANCE

INTRODUCTION

CLINICAL PROFILE AND SEVERITY OF STROKE IN DIABETIC AND NON-DIABETIC PATIENTS

INTRODUCTION

Stroke which is defined as abrupt onset of a neurological deficit that is attributable to a vascular cause, is the major cause of death worldwide. Stroke is the second most common cause of death and also the leading cause of long-term disability worldwide[1] . Various risk factors are known to increase the liability of stroke and its outcome, of which the most important being Diabetes, Hypertension, Smoking and Dyslipidemia. Diabetes mellitus is a group of metabolic disorders with vascular complications.

Diabetes mellitus is a well established independent risk factor for ischemic stroke with an overall risk of 2.5 times higher than in non-diabetic patients and is associated with greater in- hospital mortaliy and morbidity in both ischaemic stroke and intracerebral haemorrhage. In our country the estimated prevalence rate of stroke range, 84-262 per lakh population in rural and 334-424per lakh population in urban areas[2]. The incidence rate is 119-145per lakh population based on the recent population based studies[2]. This study mainly focused on severity of stroke in diabetic patients who admitted in tirunelveli medical college hospital.

The modifiable risk factor like systemic hypertension ,diabetes mellitus , life style factors such as smoking , excessive alcohol intake , stress , high fat diet ,sedentary life style increase the incidence of stroke.

The increasing number of diabetic patients in our country increases the number of noncommunicable disease among our population. Diabetes mellitus alters the lipid metabolism and promotes the formation of atheroma. Also the diabetic partients has other associated risk factors more commonly than non diabetics. All these factors increases the incidence and severity of cerebrovascular disease among diabetes patients.

AIM OF THE STUDY

Aim of the study:

1. To study the presentation of symptoms of stroke in diabetic patients.
2. To study the relationship between hyperglycemia on admission and severity of stroke in diabetic patients.
3. To study the severity of symptoms and signs of stroke in diabetic and non- diabetic patients.

Review of Literature

Review of literature

BLOOD SUPPLY OF BRAIN :

Brain gets its arterial supply from 2 pairs of vessels. The vertebral which forms the posterior circulation and internal carotid arteries which forms the anterior circulation. Both are interconnected in cranial cavity and produce circle of willis.

Anterior cerebral circulation

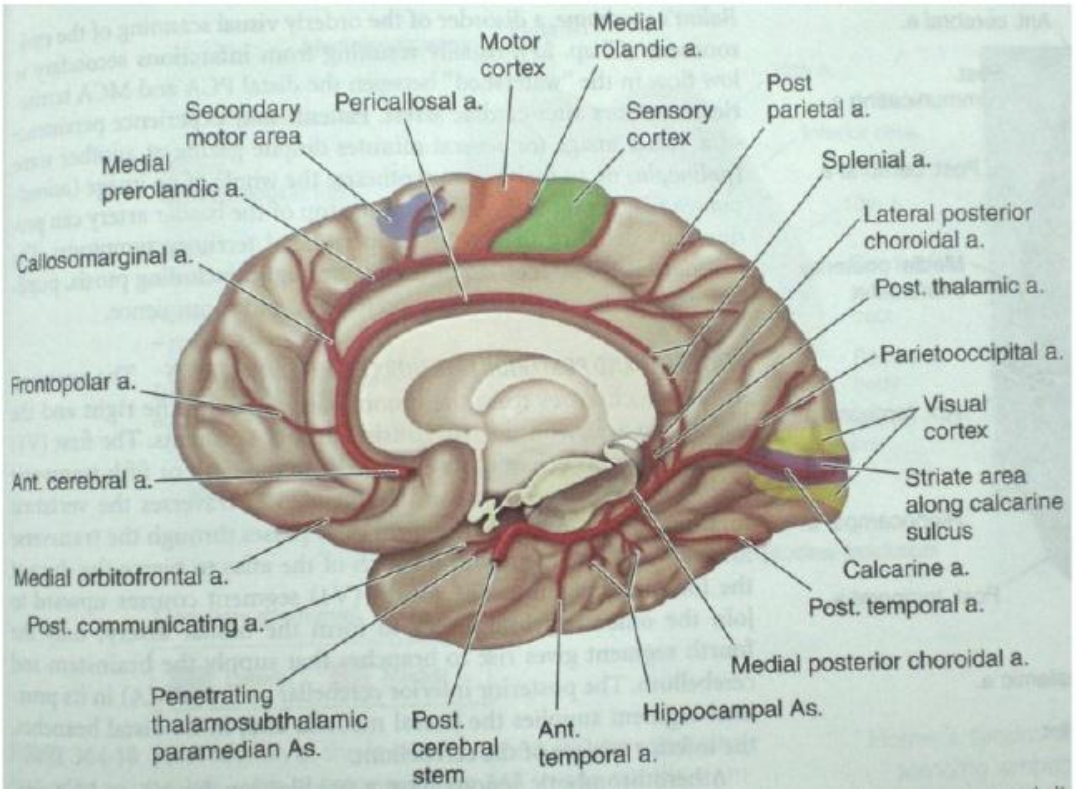
The Internal carotid artery is the branch of common carotid artery. They proceed superiorly enter in to the cranial cavity through carotid canal. By entering the cranial cavity each of the internal carotid artery gives off , the anterior cerebral artery , the middle cerebral artery and the posterior communicating artery , the ophthalmic artery.

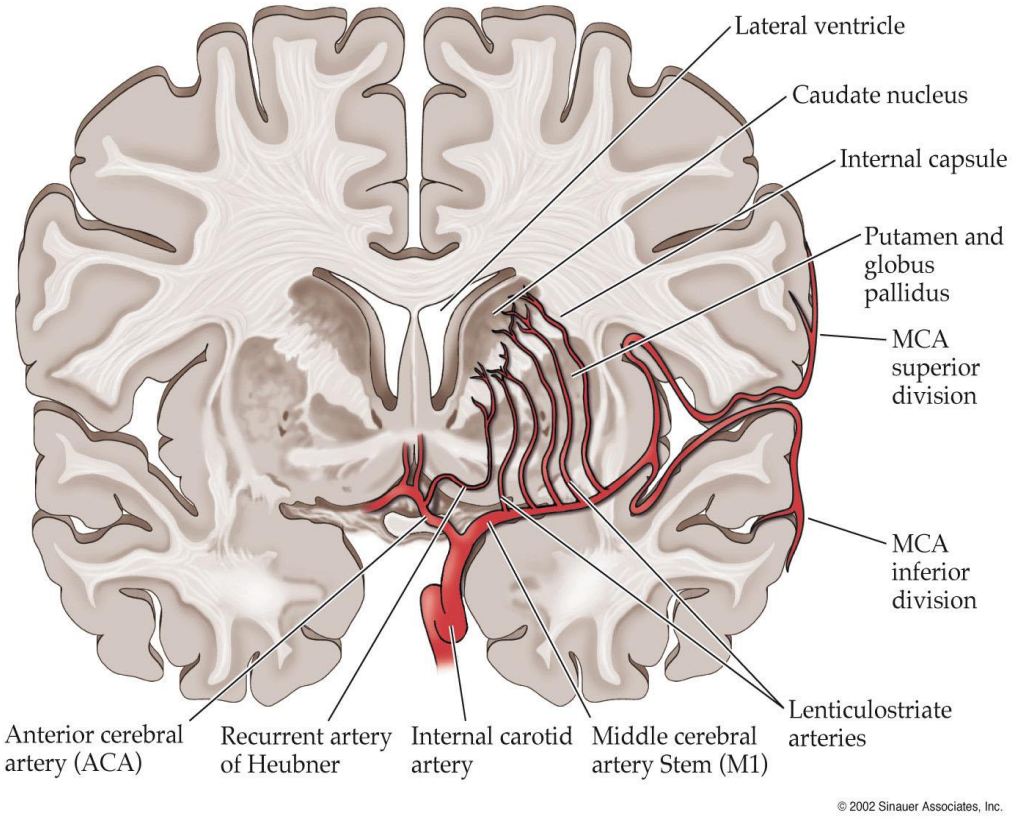
Anterior cerebral artery supplies the entire medial surface of cerebral hemisphere including a strip of cortex for about 2 cm along the superolateral surface and medial half of orbital surface except the occipital lobe. Recurrent artery of heubner is an important branch of ACA and its involvement causes faciobrachial monoplegia.

Middle cerebral artery supplies the entire lateral surface of cerebrum including lateral half of orbital surface except, frontalpole and a strip of cortex for about 2cm along superolateral surface of frontal lobe , Medial half of orbital surface , lower temporal and occipital pole.

Posterior cerebral artery supplies the medial surface of temporal and occipital lobes and their tentorial surface. It also supplies cerebellum , medulla , pons , midbrain , subthalamus and thalamus.

Branches and distribution of ANTERIOR CEREBRAL ARTERY and some branches of POSTERIOR CEREBRAL ARTERY





Above picture shows BLOOD SUPPLY BY MCA

Posterior cerebral circulation:

Vertebral arteries :

Vertebral artery arises from first part of subclavian artery. Enters the cranial cavity through foramen magnum, each vertebral artery gives small meningeal branch. Vertebral artery gives 3 additional branch,

1. Anterior spinal artery
2. Posterior spinal artery
3. posterior inferior cerebellar artery.

Vertebral artery on each side joins to form basilar artery. Its branches in a caudal to rostral direction anterior inferior cerebellar arteries, several small pontine arteries,superior cerebellar arteries.basilar artery bifurcates into posterior cerebral arteries.

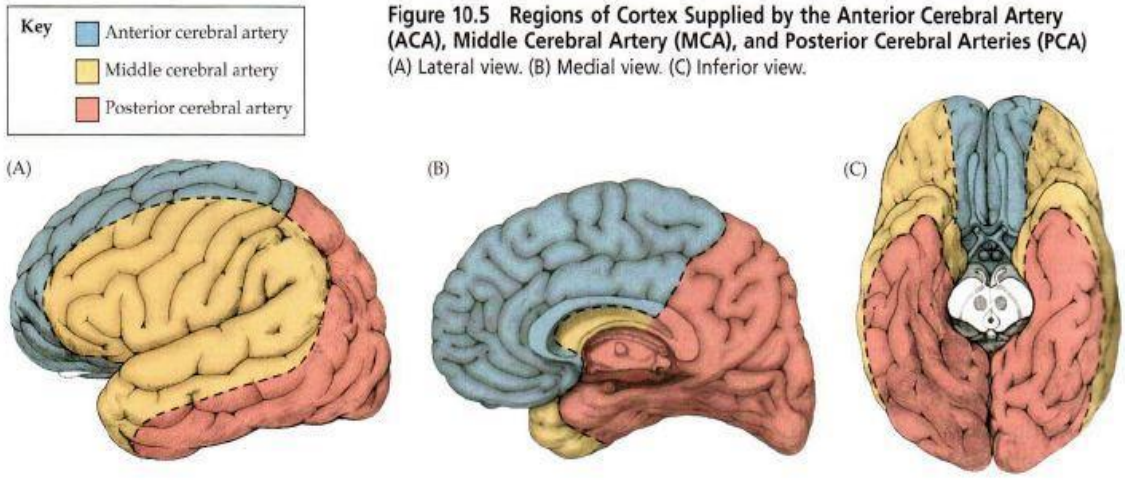
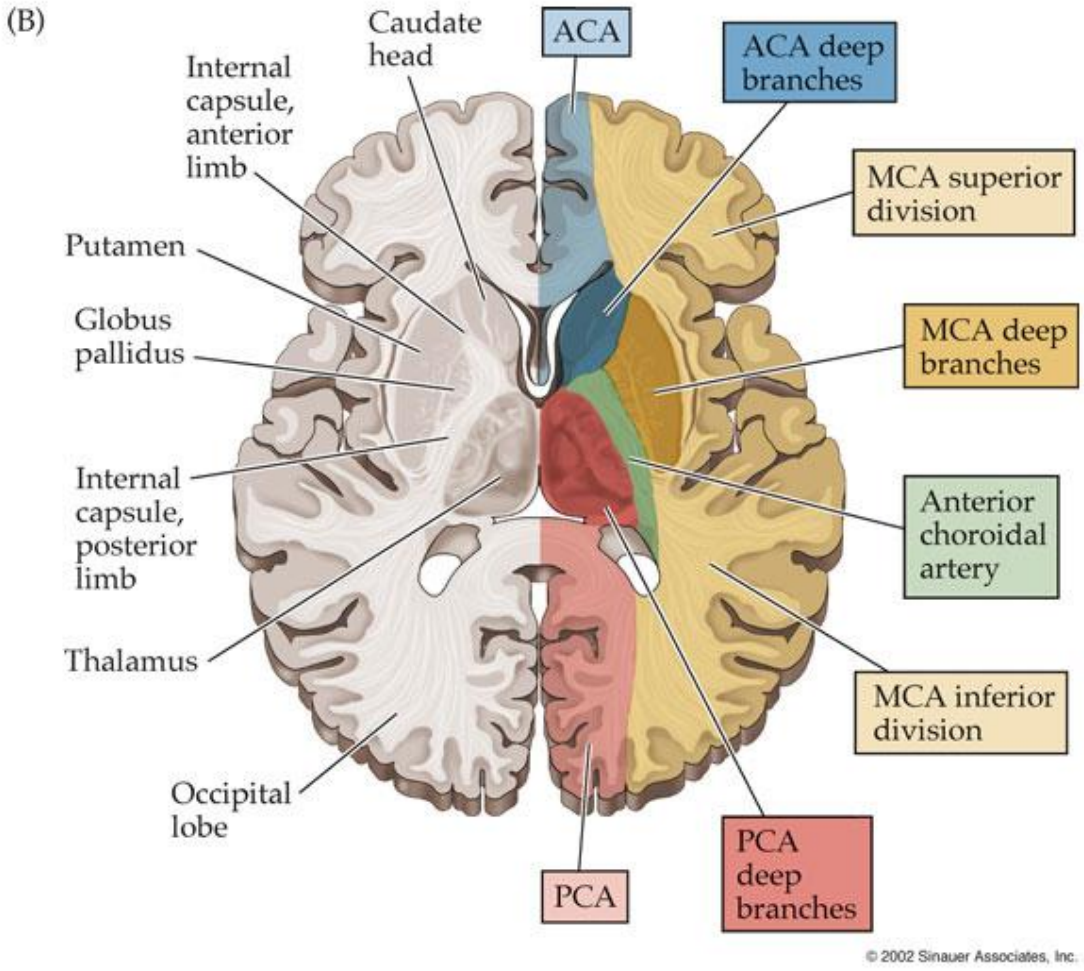


Figure 10.5 Regions of Cortex Supplied by the Anterior Cerebral Artery (ACA), Middle Cerebral Artery (MCA), and Posterior Cerebral Arteries (PCA)
(A) Lateral view. (B) Medial view. (C) Inferior view.


(B)
Caudate head
ACA
ACA deep branches
Internal capsule, anterior limb
MCA superior division
Putamen
Globus pallidus
MCA deep branches
Internal capsule, posterior limb
Anterior choroidal artery
Thalamus
MCA inferior division
Occipital lobe
PCA deep branches
PCA
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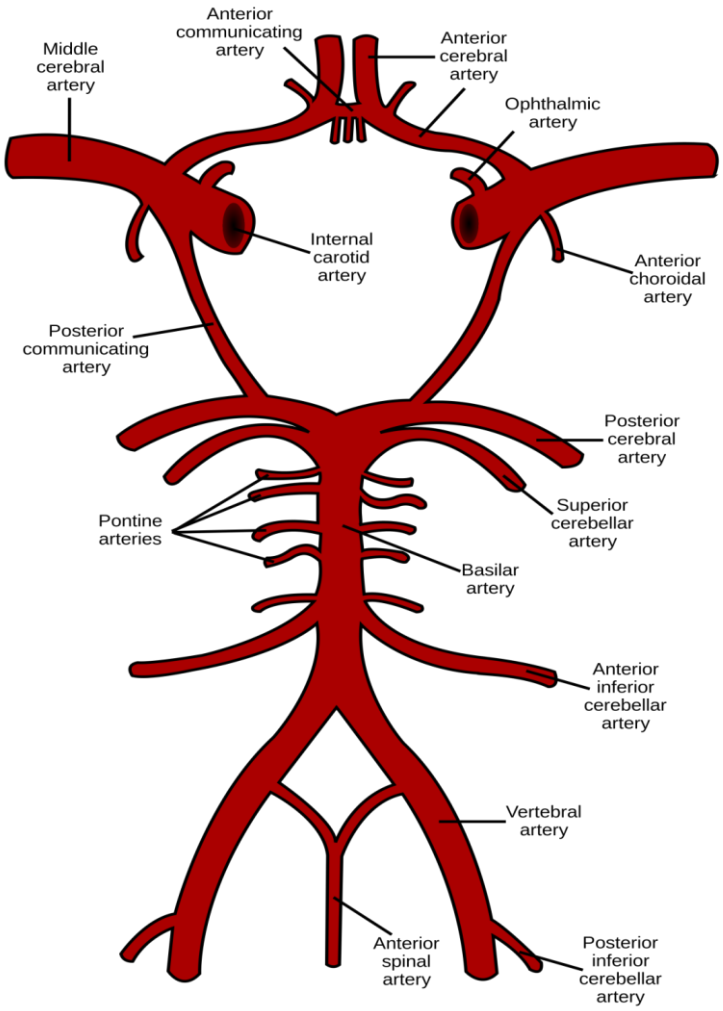
CIRCLE OF WILLIS:

Cerebral arterial circle is formed at the base of the brain by the interconnecting vertebrobasilar and internal carotid systems of arteries. This interconnections achived by an anterior communicating artery which interconnects left and right anterior cerebral arteries, 2 posterior communicating arteries one on each side connects posterior cerebral artery with the internal carotid artery.



INTERNAL CAPSULE:

The anterior limb of internal capsule is bounded medially by the head of the caudate nucleus and laterally by the lentiform nucleus. Genu of the internal capsule lies at the most medial edge of the globus pallidus. Posterior limb of internal capsule is bounded medially by the thalamus and laterally by the lentiform nucleus. Corticonuclear fibers which are motor fibers from the cerebral cortex to the motor nuclei of the cranial fibers pass through the genu of internal capsule. Corticospinal fibers pass through the anterior two third of the posterior limb of the internal capsule. Genu and posterior limb of the internal capsule is supplied by the striate branches of the anterior and middle cerebral arteries.

DIABETES MELLITUS:

Diabetes mellitus is a group of metabolic disorders characterised by chronic hyperglycemia associated with disturbances of carbohydrate metabolism, protein metabolism and fat metabolism due to absolute or relative deficiency of insulin secretion and/or action. Diabetes produces long term damage, dysfunction and failure of various organs especially the eyes, renal,nerves,and cardiac and blood vessels.it is appropriately described as METABOLIC AND VASCULAR DISORDER.

DIAGNOSTIC CRITERIA:

Fasting plasma glucose>/=126mg/dl(7mmol),2hour glucose after oral glucose challenge>/=200 mg/dl(11.1mmol) or HbA1c>/=6.5%, random plasma glucose >/=200mg/dl accompanied by classic symptoms of DM warrants the diagnosis of DM[3].

DIABETES RELATED COMPLICATIONS:

Micro-vascular complications:

ocular disease

- Retinopathy(non proliferative and proliferative)
- Macular edema

Neuropathy

- Sensory and motor(mono & polyneuropathy)
- Autonomic

Nephropathy

Macro-vascular complications

Ischemic heart disease

Peripheral vascular disease

Stroke

Others

Gastrointestinal(gastroparesis ,diarrhea)

Genitourinary (uropathy,sexual dysfunction)

Dermatologic, cheiroarthropathy

Infectious, periodontal disease

Cataract, glaucoma ,hard of hearing

RISK FACTORS FOR STROKE :

1. Gender :

Male gender increases the risk for stroke[4]. Men has 1.3 times higher risk for stroke than female except in the highest ages. Difference in the risk between the gender disappears at high age over 80-85 years. The gender risk is different for subarachnoid haemorrhage where the risk is higher for women.

2. Age:

Incidence of stroke increases with age in both men and women[5]. This increased stroke incidence is seen in ischaemic as well as for intracerebral haemorrhage, subarachnoid haemorrhage. The risk of stroke more than doubles with each decade of increased age after 55 years of age.

3. Ethnicity:

African origin people have higher risk than Caucasians. This can be explained by the poor management of treatable risk factors. The proportion of intracerebral haemorrhage is higher in Chinese than Caucasians. In ischaemic stroke, the prevalence of intracranial artery stenosis is more frequent in East Asians and African Americans than in Caucasians[6].

4. Genetic causes:

   cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy (CADASIL-notch-3gene), cerebral autosomal recessive arteriopathy and leucoencephalopathy (CARASIL-HTRA gene), MELAS (mitochondrial myopathy,encephalopathy,lactic acidosis, and stroke like episodes), homocystinuria, Fabry disease(alpha galactosidase gene), Ehlers–Danlos syndrome type IV,Marfan syndrome, pseudoxanthoma elasticum, HANAC (hereditaryangiopathy, nephropathy, aneurysm, and muscle cramps syndrome;*COL4A1* mutation)[7]. All these above mentioned syndrome has stroke or stroke like episode as one of the feature of the different clinical manifestation. Sickle cell anemia increases the incidence of stroke in childhood[8]. Increased transcranial ultrasound velocities of the middle cerebral artery has increased risk in sickle cell anemic children. Single nucleotide polymorphism in the chromosome 9p21 associated with ischaemic stroke[9], single nucleotide polymorphism in HDAC9 on chromosome 7p21.1 also associated with ischaemic stroke[10]. The APOE epsilon2&4 are related lobar intracerebral haenorrhage[11].

5. Diabetes mellitus: diabetes mellitus has a deteriorating effect on arterial blood vessels and it is a risk factor for ischemic stroke.

DM increases the incidence of recurrent stroke[12]. Lacunar infarcts may be more common in diabetic patients[13].

6. Hypertension:

It is the treatable risk factor for stroke. Hypertension is commonly detected in stroke patients with age less than 55 years of age. Both systolic and diastolic BP is of importance as risk factor[14]. A systolic BP increase of 20 mmHg or a diastolic BP increase of 10 mmHg more than doubles the risk of stroke death.

Previous stroke/ Transient ischaemic attack:

Previous stroke is a powerful risk factor for new stroke. Also TIA increases the risk of subsequent risk of stroke in short and long term. Higher risk among those with diabetes,age>60 years,longer duration of TIA, TIA with weakness or speech disturbance[15].

7. White matter disease:

Both periventricular and subcortical white matter hyperintensities also increase the risk of subsequent stroke, independently of the presence of silent brain infarcts[16].

8. Dyslipidemia:

Increased cholesterol levels are associated with carotid artery atherosclerosis and so increases the risk of cerebral infarcts caused by large vessel disease[17]. on the other hand low cholesterol level

increases the risk of ICH[18]. Intense lowering of cholesterol level in stroke patients slightly increases the chance of ICH.

9. Coagulation disorders: Antiphospholipid antibody and lupus anticoagulant have been associated with ischaemic stroke. A coagulation disorder causing a venous thrombosis may give rise to paradoxical embolism through patent foramen ovale.

10. Obstructive sleep apnea:

    obstructive sleep apnea may increase BP and cause obesity. Obstructive sleep apnea itself is an independent risk factor for stroke[19]. Possible mechanisms include hypercoagulability, atherosclerosis, decreased cerebral blood flow. Wake up stroke recently been linked to obstructive sleep apnea[20].

11. Renal disease: Renal increases the risk of stroke in individuals with known atherothrombotic disease. Microalbuminuria has been independently associated with stroke.

12. Atrial fibrillation, cardiomyopathies, patent foramen ovale, valvular heart disease increases the risk of stroke.

13. Life style risk factors:

a. Smoking: cigarette smoking increases the risk for ischaemic stroke also increases the risk for ICH[21]. Both passive and active smoking increases the risk. Individuals who stop their smoking reduce their risk of stroke by upto 50%.

b. Alcohol : Excessive alcohol increase the stroke risk. Individuals who do not use any alcohol may have a slightly increased stroke. It is possible that temporary heavy alcohol consumption increases the risk of immediate stroke[22].

c. Diet: Fruit and vegetables may have a protective effect against both ischaemic and haemorrhagic stroke. Individuals eating three to five servings per day had a relative risk of stroke of 0.89 compared with those consuming less fruit and vegetables[23]. Fish consumption also reduces the risk of stroke.

d. Physical activity: Physical activity decreases the risk of stroke compared with no physical activity[24]. Daily exercise of at least 30 minutes decreases the relative risk of stroke to between 0.69 and 0.74.Physical activity of at least 30 minutes three to five times per week has been recommended.

e. Obesity: body mass index of 25kg/m2 or more in men,30kg/m2 or more in women increases the risk of ischaemic stroke[25]. waist : hip ratio also has been related to increased stroke risk.

f. Hormonal therapy/OCP: OCP increases the risk of ischaemic stroke[26]; hormone replacement therapy has also been related to

increased risk of stroke. Active tamoxifen therapy increases the stroke risk.

g. Stress: self perceived psychological stress increases the stroke risk[27]. Both negative emotions and anger seem to be more common during the last 2 hours before stroke onset than during the preceding day or year.

h. Socio economic factors: low socioeconomic status is associated with increased risk of stroke[28]. Neurological deficit is more severe in low socioeconomic and mortality ratio is high in low socio economic status.

Risk factors of ICH: hypertension, diabetes, higher age, amyloid angiopathy, lower cholesterol, frequent alcohol use, patients using antiplatelets and anticoagulant medication. Cerebral microbleeds are more common in ICH than in ischaemic stroke/TIA.

Risk factors for subarachnoid haemorrhage: smoking, hypertension, female gender, increased age, alcohol consumption, ocp, genetic factors.

Risk factors for cerebral venous thrombosis: genetic or acquired, thrombophilia, haematological conditions including

polycythemia,thrombocythemia,anemia, and infection, dehydration. In female population younger than 50 years of age with CVT have risk factors like oral contraceptives, pregnancy, puerperium.

PATHOGENESIS :

Brain receives 55 mL to 70 mL of blood/100 g of brain/ min there by maintain its function normally. If the blood flow is less than 15 mL/100 g/min, the resulting ischaemia with hypoxia, when sufficiently prolonged, may cause death of neurons and glia. The mean arterial blood pressure, resistance of cerebral vessel, local metabolic products (pH, PaO2, PaCO2),together with many known factors & unknown factors, help to maintain the blood flow. The blood flow varies in different areas of the brain and autoregulation determines the regional flow to meet local metabolic needs. In regions of cerebral ischaemia, there is impaired self regulation and the microvasculature is unresponsive to pressure changes, to vasoactive agents and to other forms of stimuli. Cerebral edema develops if there is vascular leak. To protect the brain from ischaemia, several collateral pathways exist. The four major extra-cranial arteries (carotid and vertebral arteries) form good-calibre, low-resistance anastomoses at the base of brain . The

post-Willisian anastomoses further protect the brain tissue from the effects of occlusion of single cortical branches . However, in the presence of generalised arterial disease or multiple skipped stenotic lesions (atherosclerosis), anomalous or congenital variations, these collateral pathways may prove inadequate and predispose to cerebral ischaemia resulting in brain infarction

GENERAL CONSIDERATIONS :

If, for any reason, such a cardiac arrest or prolonged hypotension [systolic blood pressure (BP) falls below 70 mm Hg] occurs, brain tissue is significantly deprived of its nutrition for more than three minutes, ischaemic-hypoxic cerebral injury or infarction results. Such infarcts are either pale (ischaemic infarction in thrombosis) or may show petechial haemorrhages in the cortical mantle (haemorrhagic infarction in brain embolism). The sylvian region is commonly involved in middle cerebral artery occlusion (mid-field infarct) whereas with internal carotid artery lesions; the cerebral infarction is mostly located in the distal territory (end-field infarction). A mid-field infarct is produced by occlusion of a small penetrating vessel, and if located in the territory of major anatomical pathways, it may prove catastrophic. On the other hand, an endfield infarct resulting from occlusion of a major vessel in the neck with good collateral circulation may be asymptomatic. Obstruction to venous return can also result in

haemorrhagic infarction (e.g. cortical cerebral venous thrombosis). Acutely infarcted brain tissue is soft and swollen, frequently herniates downwards and may compress the vital centres within the brainstem, with fatal outcome. Like infarction elsewhere in the body, cerebral infarcts heal by gliosis leaving a firm scar or a cystic cavity.

AETIOPATHOLOGY AND PATHOPHYSIOLOGY :

Cerebral infarction is usually attributed to partial or total occlusion of its regional microvasculature by thromboembolism. Cerebral atheroma is by far the most common underlying intimal vascular pathology, whereas thrombosis associated with arteritis (syphilitic, rheumatic, tuberculosis) is not an uncommon aetiology for ‘strokes in the young’ in India. If reperfusion established within 3 hours the ischemic penumbra zone can be salvaged. High cytosolic calcium, Na+/k+ ATPase failure , acidosis, free radicals and other unknown factors, are theimportant factors imapair the blood-brain barrier (BBB) and microvascular function . with decreased ATP, there is delay in re-synthesis of macromolecular proteins which is important for endothelial cell structure and function. Energy failures also induce proteolysis and lipolysis, production of arachidonic acid and platelet activating factors, cell adhesion molecules, nitric oxide and free radicals, post-ischaemic hypo-perfusion or hyperperfusion injuries resulting in further neuronal

impairment .Thus, development of cerebral infarct is not merely the result of ischaemia but the end-result of several highly complex 'ischaemia-modifying factors.' For example, exposure of vascular endothelium to raised homocysteine (>100 μmol/L) levels leads to reduced nitric oxide, increased levels of adhesion molecules and expression of procoagulant factors like plasminogen activator inhibitor (PAI-1), tissue plasminogen activator (tPA), protein C (PC) and thrombomodulin (TM), which in turn promote platelet aggregation, leucocytes adhesion and thrombosis.

CLINICAL FEATURES OF STROKE :

Clinical Features

Obesity, feeble or absent peripheral arterial pulsations, vascular bruits, unequal or raised blood pressure, postural hypotension and retinopathy may be detected during general physical examination. In about 60% of the patients, prodromal warning symptoms of TIA may precede. Such episodes of TIA are usually brief (lasting for a few minutes to less than an hour) and may come singly or in successive spells over a number of hours or days or months, and leave no significant residual signs. TIAs may not be always related to posture or the level of BP, and may disappear altogether. However, in some cases (10% to 15%), an evolving or a full-blown stroke may follow the last ischaemic spell. When the stroke evolves in a stepwise manner,

('thrombosis in evolution') the symptoms may appear in each limb in succession or simultaneously. This stuttering or intermittent progression is typical of atherothrombosis. Not infrequently, the stroke may announce itself abruptly as a single major catastrophic event (accomplished infarction or completed stroke). The other clinical manifestations depend on the site of arterial occlusion.

INTERNAL CAROTID SYNDROME :

The cervical portion of the internal carotid artery near the carotid sinus is a common site for athero-stenosis and about 60% of all thrombotic lesions are located here. Often, these lesions may be asymptomatic because of collateral anastomoses (external carotid-ophthalmic anastomoses or from superficial and deep cervical anastomoses or from the opposite carotid artery through the anterior segment of the Circle of Willis). Warning symptoms precede a major ictus in nearly 50% of the subjects. Such symptoms include brief episodes of confusion, with speech difficulty (aphasia, dysarthria, dyslexia), sensory paraesthesia with or without motor weakness of the opposite side. Ipsilateral amaurosis fugax (transient monocular blindness), fleeting or semi-permanent, alternating with or accompanied by a contralateral hemiplegia or sensory deficit, is pathognomonic of carotid artery syndrome; however, these events occur in only 15% to 20% of the patients.

The clinical manifestations of acute carotid artery occlusion are almost indistinguishable from those of middle cerebral syndrome. Feeble internal carotid or superficial temporal artery pulsations, dilated pupil and poorly pulsating retinal vessels on the side of the suspected carotid lesion and ocular or cervical bruits on the ipsilateral side may suggest the correct diagnosis. Carotid duplex Doppler sonography and angiography are helpful in defining the extent and degree of stenosis. In patients with an old or silent occlusive carotid artery lesion on one side, a new lesion on the opposite side may prove catastrophic. Here, the physical finding of bilateral hemiplegia (quadriplegia) with coma can be mistaken for basilar artery syndrome.

ASYMPTOMATIC CERVICAL BRUIT :

A carotid bruit may be heard in the neck in about 5% of asymptomatic elderly patients (55 years to 80 years). Unless haemodynamically significant, it is very difficult to correlate the mere presence of a bruit with a subsequent TIA or stroke in that territory. The role of prophylactic endarterectomy to prevent a future stroke (estimated at 6% within next 3 years) has not been established by clinical trials. In such cases antiplatelet therapy may be prescribed.

MIDDLE CEREBRAL SYNDROME :

The cortical branches supply lateral surface of the cerebral hemisphere, except for the regions supplied by the anterior and posterior cerebral arteries. The areas of supply include the sensory-motor cortex, the motor and sensory speech centres, auditory area and optic radiation. The penetrating branches (lenticulo-striate arteries) supply the putamen, globus pallidus, genu and posterior limb of the internal capsule. The clinical picture of middle cerebral artery occlusion is variable. Contralateral hemiplegia, hemianaesthesia with or without homonymous hemianopia and aphasia (dominant hemisphere) are common manifestations. Occlusion of the superior division presents as contralateral hemiparesis with sensory deficit and expressive aphasia (Broca's aphasia), whereas Wernicke's aphasia (sensory aphasia) is frequent with lesions of the inferior division of dominant side. Monoplegic symptoms with lesion of single cortical branches are not uncommon. Occlusion of penetrating branches (lenticulo-striate arteries) has been repeatedly blamed for a dense sensorimotor hemiplegic syndrome ('capsular-hemiplegia'), but significant sensory loss seldom occurs with such a lesion and 'pure motor hemiplegia' is common.

ANTERIOR CHOROIDAL SYNDROME :

This artery supplies the posterior limb of the internal capsule, which carries the corticospinal and sensory fibres for the contralateral limb. This syndrome, which represents a true ‘capsular-hemiplegia’ (dense hemiplegia, hemianaesthesia and homonymous hemianopia), is rare.

ANTERIOR CEREBRAL SYNDROME :

The cortical branches mainly supply the medial superior surface of the frontal lobe and the parietal lobe up to the paracentral lobule. The penetrating branches supply the anterior limb of the internal capsule and part of the head of caudate nucleus.

An anterior cerebral artery occlusion proximal to the anterior communicating artery, in subjects with a symmetrical Circle of Willis, is frequently asymptomatic. Occlusion, distal to the anterior communicating artery, manifests itself by a sensorimotor paralysis of the opposite lower extremity with mild weakness of the opposite shoulder. Mental changes, ictal and urinary incontinence, gait disturbances, apraxia, grasp and sucking reflexes may accompany the above findings.Occlusion of an unpaired anterior cerebral artery (supplying both the hemispheres) results in a cortical type of paraplegia, with sphincter incontinence and a mental state in which the patient is alert but mute (akinetic mutism). Aphasia and hemianopia are never seen. Occlusion of the penetrating branches and of the Heubner’s artery is frequently blamed for ataxic tremors of the contralateral limbs

(frontal ataxia). Apraxias, idiomotor dyspraxia of the limbs and gait may also be present.

POSTERIOR CEREBRAL SYNDROME :

This artery supplies the medial and inferior aspects of the occipital and temporal lobes. Its branches also supply the midbrain, cerebral peduncle, most of the thalamic and subthalamicregions. Embolic occlusion of the posterior cerebral arteries is not uncommon. Contralateral homonymous hemianopia is a significant finding and this results from infarction of the primary visual area (calcarine cortex); the central vision is frequently spared, even in cases with bilateral disease (gun-barrel vision). Other manifestations of visual dysfunction include illusory or distorted vision, visual-object agnosia and various forms of dyslexia without dysgraphia. The pupillary reflexes are well preserved. Contralateral hemiplegia from lesion of the cerebral peduncle (peduncular hemiplegia) and thalamic syndrome (Dejerine-Roussy syndrome) may also be present. In the thalamic syndrome, there is varying degree of sensory loss to all modalities and spontaneous burning or agonising pains are frequent (analgia dolorosa). Memory loss (amnesia) suggests lesion of the medial temporal cortex. Contralateral involuntary choreoathetosis or ataxic tremors are rarely observed.

VERTEBRO-BASILAR SYNDROME :

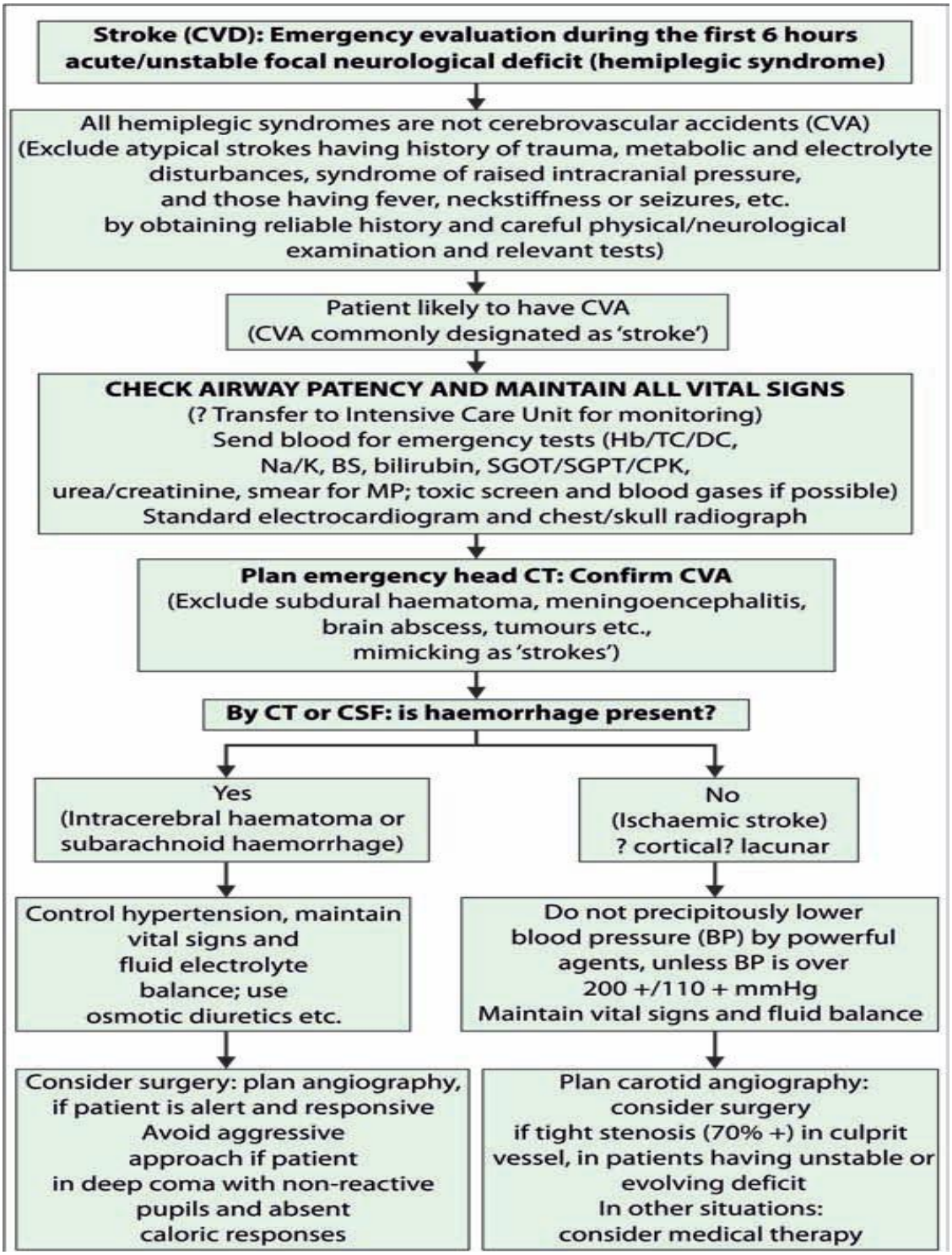
After traversing through the bony vertebral canals both vertebral arteries unite intra-cranially to form the basilar trunk. Their short paramedian and long circumferential branches supply the entire brainstem, cerebellum and the vestibular apparatus. TIA manifest as episodes of vertigo, dizziness, diplopia, dysarthria, dysphasia, incoordination of gait and limbs and bilateral signs of sensorimotor deficit.

Occipital headaches may be present. Ipsilateral 3rd nerve palsy with contralateral hemiplegia (Weber's syndrome) or with crossed cerebellar ataxia (Claude's syndrome) is diagnostic of midbrain localisation. Homolateral paralysis of the 7th nerve with contralateral hemiplegia and hemianaesthesia (Millard-Gubler syndrome) is suggestive of a pontine lesion. Palatal paralysis and ataxia of limbs, with impairment of posterior column sensation on same side of the body accompanied by diminution of pain and thermal sense on the opposite limbs (Wallenberg's syndrome) indicate lateral medullary infarction from a distal vertebral artery lesion. Quadriplegia with bilateral conjugate, lateral gaze palsy and 'mute state' with fully preserved consciousness has been described ('locked-in syndrome') in infarction of the basis pontis (sparing the tegmentum), from a mid-basilar occlusion. Occlusion of isolated cerebellar branches may produce dizziness,

nausea, vomiting, nystagmus and appendicular or truncal ataxia without sensorimotor deficit in any limb. Such a syndrome should be differentiated from cerebellar haemorrhage where emergency surgical decompression may prove life-saving.

AORTIC ARCH SYNDROME :

The intriguing clinical syndrome is characterised by diminution or absence of the arterial pulsations in the vessels of the arms and the neck; the seat of the disease, irrespective of its aetiology, being located near the origins of the great vessels arising from the aortic arch. Several aetiological factors like congenital anomalies, trauma with or without aneurysm, chronic dissecting aneurysm, mediastinal tumours, thrombophilia syphilitic aortitis and atheromatosis appear to be one of the causes of this syndrome, and that an arteritis of undetermined origin is responsible for a good number of female cases. Likewise, it has been a prevalent impression that nearly all the cases of aortic arch syndrome reported from India are a form of arteritis of rheumatic, syphilitic or undetermined origin. It has now been appreciated that the primary lesion in such cases, particularly in men may not always be an arteritis.



STROKE MANAGEMENT OUTLINE

CLINICAL FEATURES OF ICH :

The focal signs and symptoms accompanying ICH reflect the location of the haemorrhage and are indistinguishable from ischaemia occurring in the same vascular territory. Lobar haemorrhages frequently produce contralateral weakness or sensory loss, language disturbance, hemianopia or lesser field disorders, and parietal lobe signs. Their relationship to the cortex makes them more likely to be complicated by seizures. Seizure are more frequent in ICH, occurring in as many as a quarter of individuals, although they are also seen in about 5% of patients with ischaemia, particularly early after onset. Occasionally, very small haematomas present with symptoms indistinguishable from transient ischaemic attacks (TIAs). However, majority of ICHs cause complete stroke. Putaminal haemorrhages typically cause contralateral hemiparesis with variable degrees of sensory loss, ataxia and with larger haematomas, a homonymous hemianopia. Thalamic haemorrhages can result in contralateral sensory loss and weakness, while if they extend to or compress the superior midbrain, they may result in depressed signs. Pontine haemorrhages often result in reduced consciousness, pinpoint pupils, bilateral weakness and pontine cranial nerve dysfunction, and may be severely disabling. Cerebellar haemorrhages are particularly important to identify clinically as they may require surgical intervention, which can be life-saving. The onset can be deceptive, with initial nonspecific brainstem symptoms (e.g.

vertigo or double vision), followed a few hours or even days later by progressive clinical features, including gait, trunk or limb ataxia, nystagmus, headache, vomiting and coma from brainstem compression. Hemiparesis will be rare. The clinical course of cerebellar haemorrhage can be difficult to predict at onset. An abrupt deterioration may occur after a period of clinical stability. Moderate degrees of quadrigeminal cisternal compression predicts poor outcome unless the haematoma is evacuated early in the course. Severe mass effect on the cistern carries a poor prognosis.

MIDDLE CEREBRAL ARTERY INFARCT:

It can present as cortical or deep infarct or both.

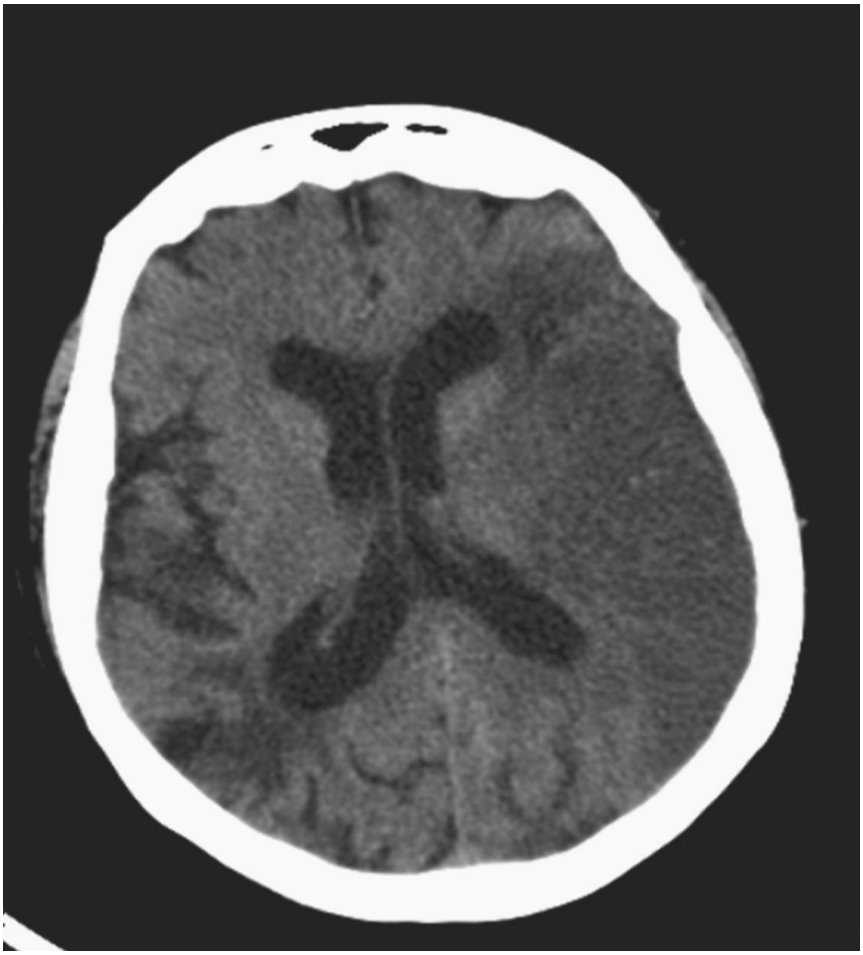
SUPERFICIAL MIDDLE CEREBRAL ARTERY INFARCTS:

Left superficial middle cerebral artery infarct associated with language disturbance and right superficial middle cerebellar artery associated with neglect.

LARGE MIDDLE CEREBRAL ARTERY INFARCT :

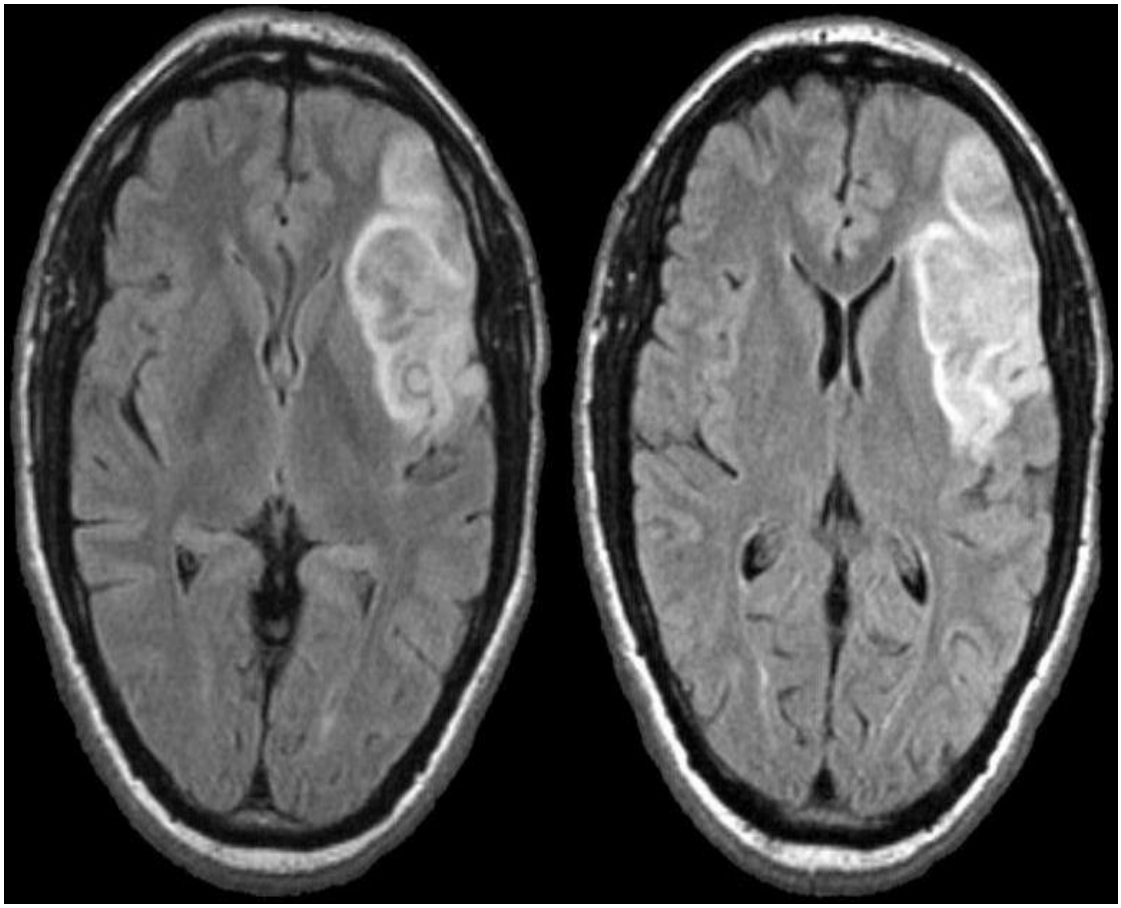
Large infarcts cover occlusion of superficial branch of middle cerebral artery and deep branch of middle cerebral artery .They have an unfavourable prognosis as there is large area of infarct and produce a detrimental neurological deficit , which includes loss of consciousness.

Large left middle cerebral artery infarct



ANTERIOR OR SUPERIOR DIVISIONS OF MIDDLE CEREBRAL ARTERY INFARCT:

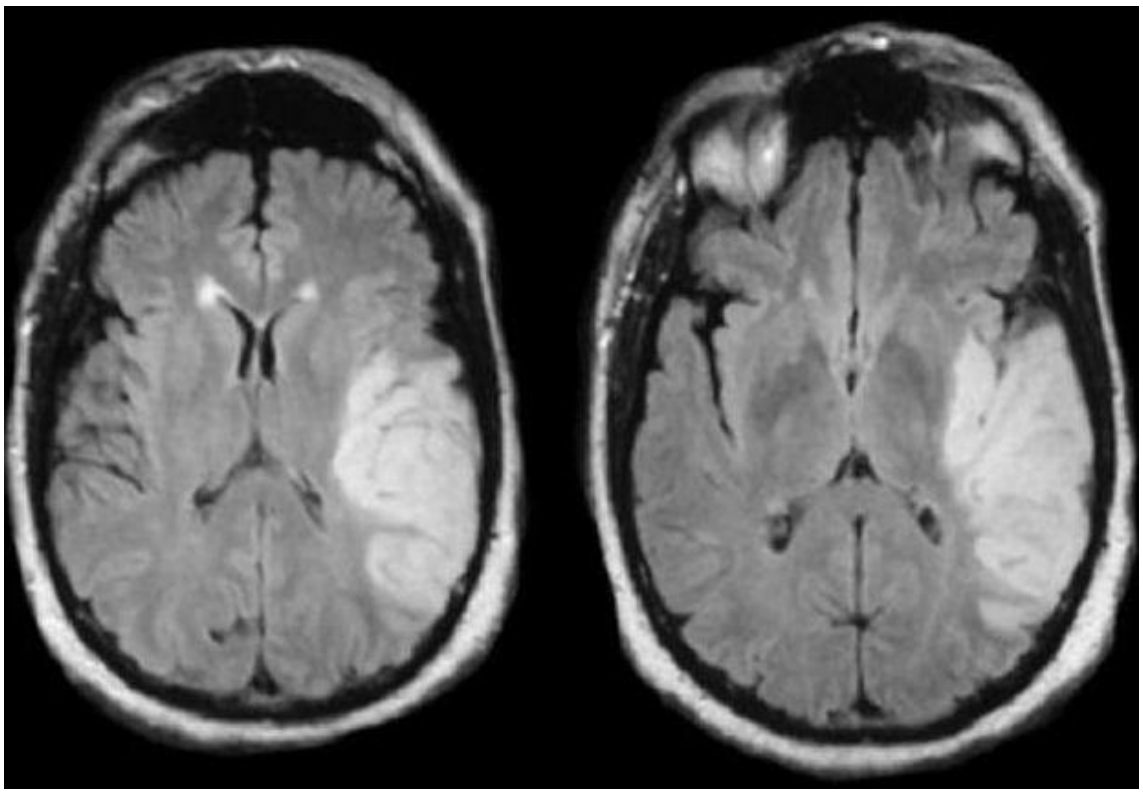
It causes opposite side hemiparesis , loss of sensation in contralateral side, gaze preference towards the side of lesion. Left side infarcts produce aphasia, buccofacial apraxia. Right side infarcts produce neglect with anosognosia.



The above picture : MRI axial flair shows left anterior branch of MCA infarct.

MIDDLE CEREBRAL ARTERY POSTERIOR OR INFERIOR DIVISION INFARCT:

It produces sensory loss ,hemianopia with no or mild motor involvement. In left hemispheric infarct fluent aphasia predominates. In right posterior branch infarct produce neglect, altered sensorium .



MRI axial flair : left posterior branch of MCA infarct

CORTICAL BRANCH SYNDROME:

Orbitofrontal infarct produce a frontal syndrome. Left prefrontal infarcts produce transcortical aphasia, right prefrontal infarcts produce motor neglect.

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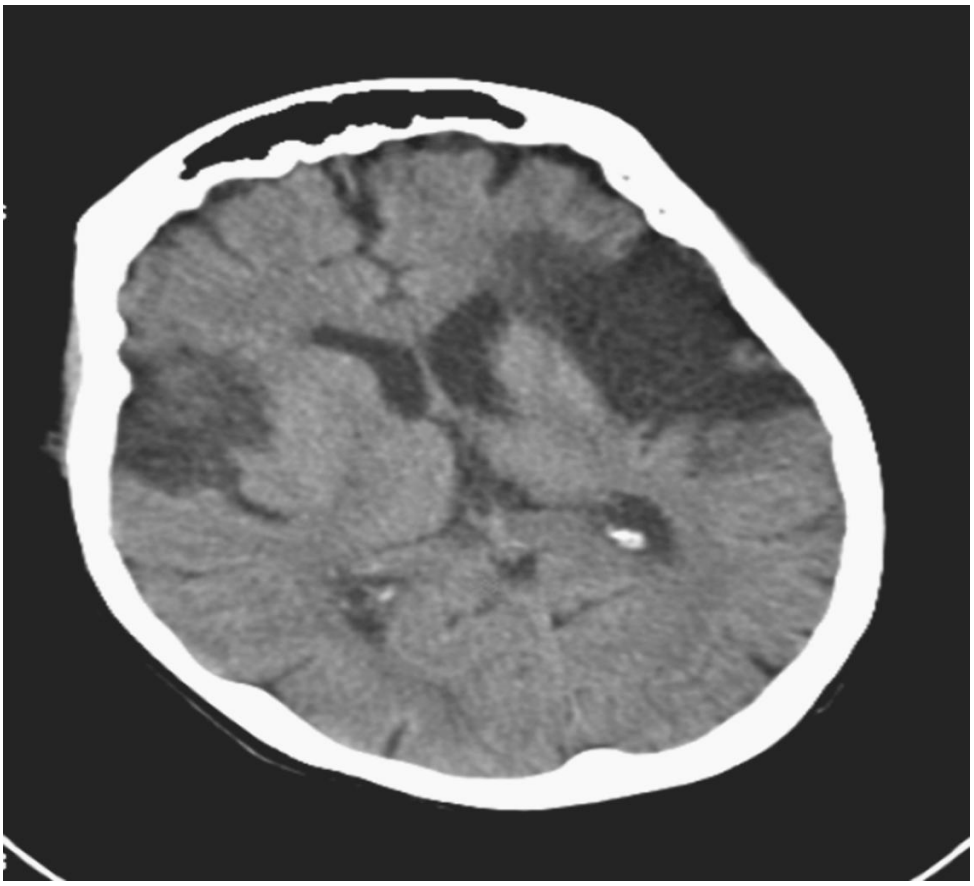
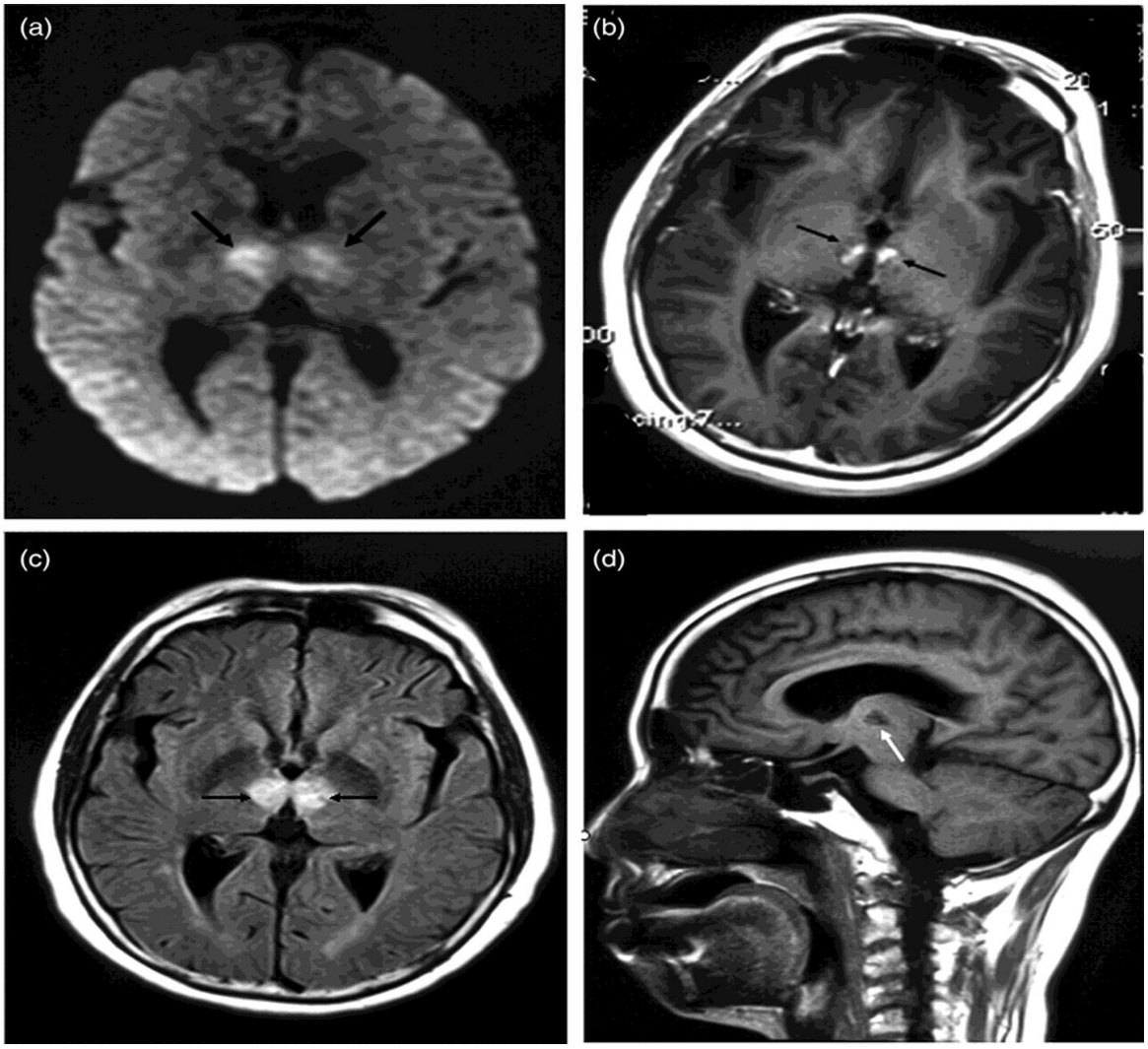
Central sulcus or rolandic artery territory infarcts produce opposite side motor weakness and opposite side sensory loss.

Bilateral central sulcus infarct



This above picture shows bilateral central sulcus infarct, which causes bilateral motor weakness and sensory disturbances.

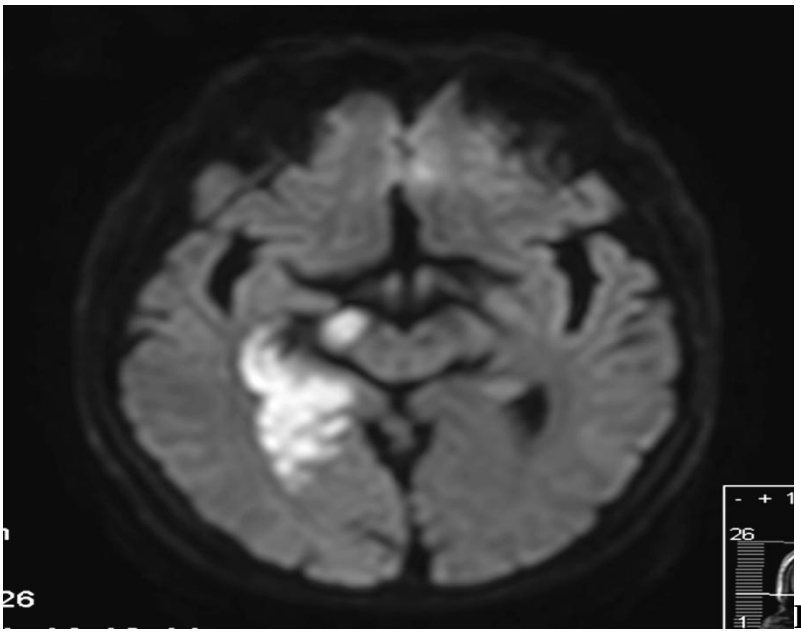
BILATERAL THALAMIC INFARCT :



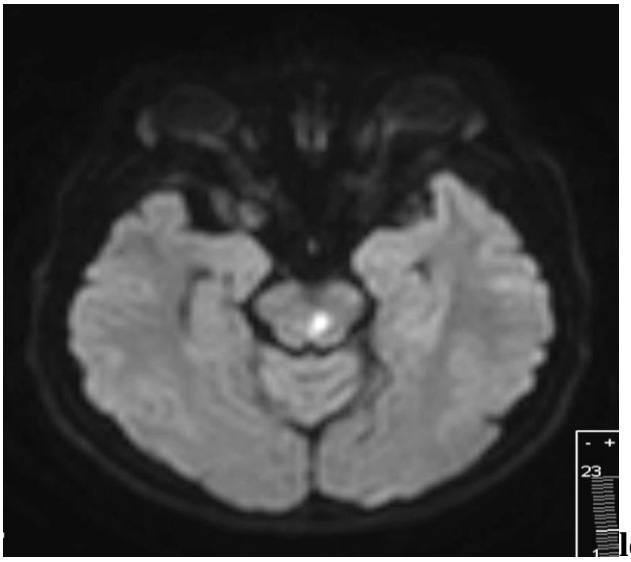
When bilateral thalamus supplied by single artery that originates from p1 segment occlusion.

POSTERIOR CEREBRAL ARTERY INFARCTS :

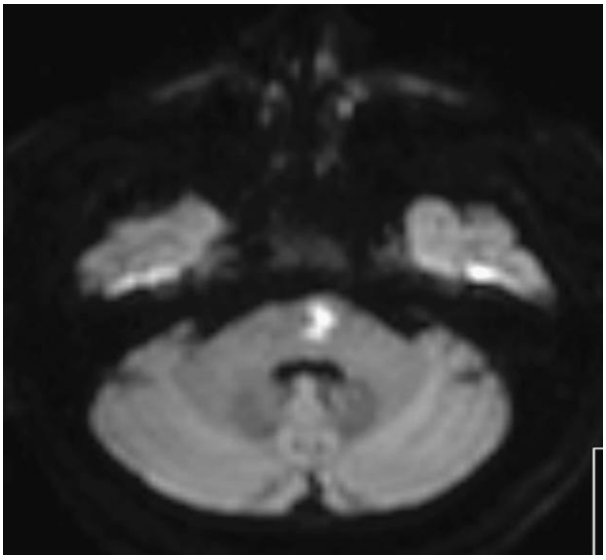
Visual field defects, including hemianopia are the main clinical findings of PCA infarcts. Headache, generally unilateral, is rather frequent .



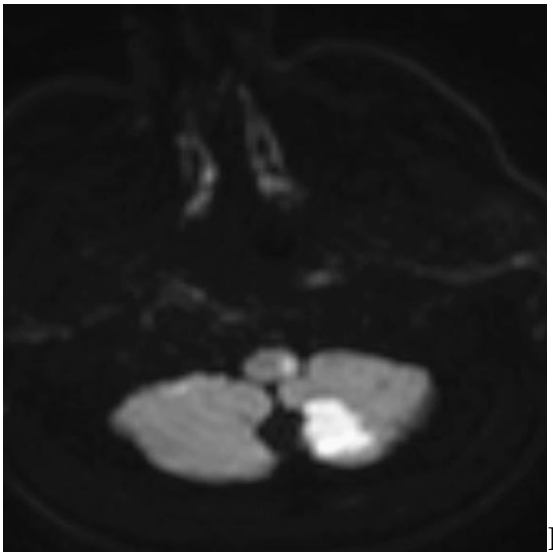
Rt pca territory infarct

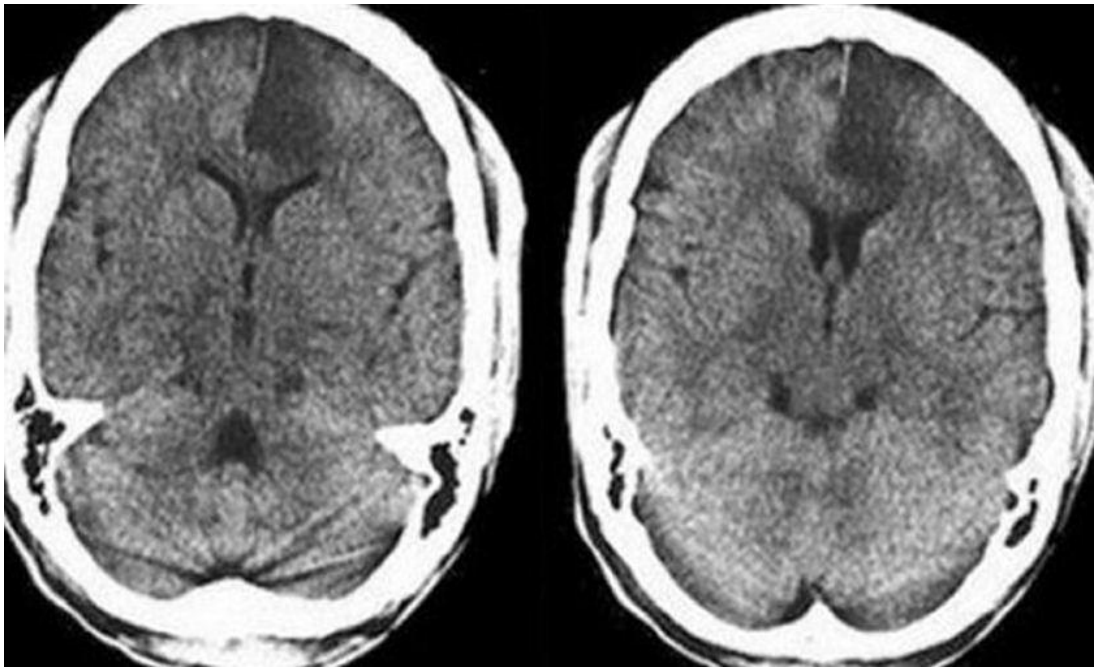


left paramedian tegmentum infarct



Left paramedian pontine infarct.


Left PICA infarct



Left anterior cerebellar artery territory infarct.

THE OXFORDSHIRE COMMUNITY STROKE PROJECT (OCSP) CLASSIFICATION :

Oxfordshire Community Stroke Project Classification (OCSP)

Total Anterior Circulation Stroke (TAC)	All of • Hemiplegia contralateral to the cerebral lesion, usually with ipsilateral hemisensory loss • Hemianopia contralateral to cerebral lesion • New disturbance of higher cerebral function (dysphasia, visuospatial)
Lacunar Stroke (LAC)	• Pathological definition • Occlusion of a single deep (LS) perforating artery • 5% can be due to haemorrhage • Occurs at strategic sites • More likely seen on MRI than CT scan • Classical lacunar syndromes correlated with relevant lacunes at autopsy
Partial Anterior Circulation Stroke (PAC)	Any of • Motor / sensory deficit + hemianopia • Motor/sensory deficit + new higher cerebral dysfunction • New higher cerebral dysfunction + hemianopia • New higher cerebral dysfunction alone • A pure motor/sensory deficit less extensive than for LAC (eg. confined to one limb, or to face and hand but not to whole arm)
Posterior Circulation Stroke (POC)	Any of • Ipsilateral cranial nerve palsy (single / multiple) with contralateral motor and/or sensory deficit • Bilateral motor and/or sensory deficit • Disorder of conjugate eye movement (horizontal/vertical) • Cerebellar dysfunction without ipsilateral long tract sign • Isolated hemianopia or cortical blindness Other signs include Horner's sign, nystagmus, dysarthria, hearing loss, etc
Code last letter as follows:	
(S) (I) (H)	Syndrome: Indeterminate pathogenesis, prior to imaging (e.g. TACS) Infarct (e.g., TACI) Haemorrhage (e.g., TACH)

THE TOAST CLASSIFICATION OF ACUTE ISCHAEMIC STROKE :

(TOAST- TRIAL OF ORG 10172 IN ACUTE STROKE TREATMENT)

Table 1 – Modified TOAST classification of ischemic stroke subtypes[8]

1. Atherosclerosis of great vessels
2. Cardioembolism (excluding cases attributed to PFO/atrial septal defects)
3. Occlusion of small vessels (lacunar)
4. IS of another etiology (defined)
5. Two or more identified causes
6. Cryptogenic ischemic stroke

PFO - patent foramen ovale; IS - ischemic stroke.

STROKE TREATMENT GUIDELINES :

ACUTE ISCHEMIC STROKE :

Includes Medical support, IV thrombolysis, endovascular revascularization, antithrombotic treatment, neuroprotection, stroke centers and rehabilitation.

MEDICAL SUPPORT :

Blood pressure lowering in ischemic stroke is indicated if there is malignant hypertension, associated acute coronary disease. Antihypertension to be used if BP > 185/110 mmhg, or when thrombolytic therapy to be administered. Lowering the BP with a beta-1 blocker is the first step to reduce cardiac work and maintain bloodpressure. Fever control with antipyretics and surface cooling. Serum glucose should be monitored kept at <180 mg/dl. The larger the size of infarct chance of developing cerebral edema is more and should be treated with iv mannitol. Prevetntive measures regarding infection , deep venous thrombosis should be taken.

IV THROMBOLYSIS :

INDICATION :

1. Definitive Clinical diagnosis of stroke.
2. Onset of ypmtoms to the time of administration of drug </= 4.5 hours.
3. No evidence of haemorrhage on CT brain.

4. Age >/= 18 years
5. Informed consent from patient or surrogate.

CONTRAINDICATIONS :

1. Sustained blood pressure > 185/110 mmhg despite treatment
2. Previous episode of stroke within 3 months; head injury within 3 months; previous episode of intracranial hemorrhage.
3. Minor stroke symptoms or rapidly improving symptoms
4. Platelet <100000;HCT<25%;glucose >400mg/dl
5. GI bleeding in preceding 21 days
6. Heparin use within 48 hours and prolonged PTT or elevated INR.
7. Major surgery in preceding 14 days.
8. Recent MI
9. Coma or stupor

ADMINISTRATION OF rtPA :

- Iv rtPA 0.9 mg/kg (maximum dose of 90 mg) iv as 10% 0f total dose administered bolus, followed by remainder of total dose administered over 1 hour.
- Frequent BP monitoring.
- No other antithrombotic treatment for 24 hours.
- Avoid urethral catheterization for 2 hours
- If there is decline in neurological condition, stop the infusion and reimage the brain emergently, give cryoprecipitate if necessary.

ENDOVASCULAR REVASCULARIZATION :

PROACT 2 trial found benefit for intra arterial prourokinase in acute MCA occlusions upto the 6^{th} hour following onset of stroke.

Endovascular mechanical thrombectomy used as an alternative or adjunctive treatment of acute stroke in patients who are not eligible for or who have contraindications or who have failed thrombolytic therapy.

ANTITHROMBOTIC TREATMENT :

According to IST and CAST trial aspirin use within 48 hours of symptoms reduced the stroke recurrence risk and mortality minimally.

ANTICOAGULATION :

A recent meta-analysis os all forms of heparin found no benefit in acute ischemic stroke.

REHABILITATION :

Early physical , occupational , speech therapy. Preventing the complications like pneumonia, urinary tract infection, deep venous thrombosis , bed sore.

TREATMENT OF INTRACEREBRAL HEMORRHAGE :

Any identified coagulopathy should be treated. Prothrombin complex concentrate, fresh frozen plasma can be used. When ICH is associated with thrombocytopenaia fresh platelet transfusion is

indicated. Adequate BP control. For cerebellar hemorrhages neurosurgeon should be consulted immediately regarding evacuation. Most cerebellar haematoma more tha 3 cm requires surgical evacuation. Less than 1cm surgical evacuation is unnecessary.1-3 cm requires careful observation.

PREVENTION :

1. Adequate BP control in chronic hypertension
2. Avoid excess alcohol intake.
3. Discontinuing illicit drugs like cocine and amphetamines.
4. In amyloid angiopathy avoid oral anticoagulants. Antiplatelets can be used if there is an indication.

STROKE SCALES:

Stroke scales are used to assess the severity of stroke at the time of admission, and predict the outcome. They can be either Clinico-metric scales or functional impairment scales or handicap scales. National institute of health stroke scale, Scandinavian stroke scale, Canadian neurological scale, European stroke scale, Mathew stroke scale, orgogozo stroke scale, hess and hunt scale, oxfordshire community stroke project classification(bamford) are used for acute assessment of stroke. Modified rankin stroke scale, Lawton IADL scale, berg balance scale, stroke specific quality of life measure(ss-qol) are functional assessment scales. Barthel index, glascow outcome scale, functional

independence measurement, health survey sf-36 & 12 are outcome assessment scales. An accurate and valid, reproducible stroke assessment scale with less interpersonal variability is essential to quantify the stroke severity.

Among these National institute of health stroke scale is the most commonly used acute stroke assessment scale and it is most popular among neurologist. Previous stroke trial results, which are the basis for stroke management are based on the initial NIH stroke scale.NIH stroke scale has less interpersonal variability,easy to assess,better reproducibility.

NATIONAL INSTITUTE OF HEALTH STROKE SCALE :

NIH stroke scale is initially developed as a research tool, now it is widely used as acute stoke assessment and predict the outcome of acute stroke patients and determine appropriate treatment for acute stroke patients. The NIH stroke scale is the better predictor of the size of the lesion and so the better measurement of stroke severity. It is a 15 item neurologic assessment scale, ratings of each item are scored with 3 to 5 grades with 0 being the normal.NIH stroke scale comprises of ,

1. Level of consciousness which has three parts. LOC responsiveness(score 0-3), LOC questionnaire (0-2),LOC command (0-2).

2. Horizontal eye movement / best gaze (0-2).
3. Visual field test (0-3).
4. Facial palsy (0-3).
5. Motor arm right (0-4), motor arm left (0-4).
6. Motor leg right (0-4), motor leg left (0-4).
7. Ataxia limb (0-3).
8. Sensory (0-2).
9. Best language (0-3).
10. Dysarthria/ speech (0-2).
11. extinction and inattention (0-2).

NIH stroke scale total score	Severity of stroke
0	No stroke symptoms
0-4	Minor stroke
5-15	Moderate stroke
16-20	Moderate to severe stroke
21-42	Severe stroke

With minimum score being 0 (no stroke symptoms), maximum score is 42 (severe stroke).

NIH STROKE SCALE USAGE :

1. To assess the severity of stroke at presentation

2. one of the assessment tool for eligibility for tPA

3. strong predictor of outcome, (NIHSS less than 6 indicates better chances of good recovery,more than 16 indicates worst prognosis),provides substantial prognostic information regarding 30 day mortality risk in acute stroke patients.

4.able to predict the stroke location, volume of brain damaged.

The incidence and prevalence of stroke in adults with diabetes is high, the risk of death also higher than people of similar age without diabetes. It is about 30% of stroke patients are diabetics and 16% of diabetics have stroke. stroke severity, outcome, chances of recurrence of stroke are more for diabetic patients. Diabetic patients have more traditional risk factors than non diabetic patients[29]. So physical activity, low carbohydrate diet, low fat diet, careful monitoring of diabetes and hypertension, weight loss will reduce the severity of stroke, improve the outcome and quality of life and reduces the recurrence of stroke. Hyperglycemia on admission is an important predictor of outcome in diabetics and non diabetic patients. Complications like urinary

infection, pneumonia, seizures were more frequent in diabetic patients with stroke than non- diabetic patients with stroke.

Glucose insulin in ischemic stroke patients (GIST), stroke patients without past history of diabetes, had 21% diabetes, 37% of patients had IGT ,42% of patients had normal glucose values after 3 months of stroke[30]. According to a FINNISH study, diabetic patients have stroke mortality of 16% for men and stroke mortality of 33% for women. Nearly 50% of people with impaired glucose tolerance develop diabetes over the period of 10 years, but lifestyle changes like physical activity, diet, weight control can substantially reduce this progression. Early detection of diabetes and to control blood glucose and other risk factors are essential in stroke patient. Hyperglycemia during the acute phase of stroke worsens the outcome, probable mechanism is high lactate level in brain tissue reduces the salvage of the penumbral tissue.

Obesity increases the risk of type 2 diabetes mellitus. Type 2 diabetes mellitus is the important risk factor for the development of cardiovascular events including stroke. Type 2 diabetes not only increases the incidence of stroke events, also influences the severity of stroke and its outcome. These increased incidence and severity is attributable not only to hyperglycemia, but also to insulin resistance, inflammation and activation of coagulation cascade. Diabetic patients

who were on antihyperglycemic agents like insulin, metformin, sulfonylureas had less severe stroke than patients who are not on antihyperglycemic drugs[31]. Thiozolidinediones enhance recovery in stroke patients. In an animal study treatment with linagliptin prior to stroke increases the number of surviving neurons than glimepiride in diabetic mice. Treatment with linagliptin increases the GLP-1 level. Anti inflammatory, anti oxidant, anti apoptotic actions appear to contribute to the neuroprotective action. Treatment with linagliptin increases the bioavailability of glucose dependent insulinotrophic polypeptide, pituitary adenylate cyclase activating polypeptide, stromal cell derived factor 1a which promote synaptic plasticity,neurogenesis, neuronal differentiation and reduce the severity of stroke. Recent study shows addition of linagliptin to metformin reduces the risk of nonfatal stroke than addition of glimepiride.

By using NIH stroke scale as a predictor for care disposition among acute stroke patients on admission could reduce the costly processes, unnecessary length of stay at hospital. Atherosclerosis is the most important risk factor for stroke patients. Diabetes mellitus accelerates the atheroma formation. CVA with high NIH stroke scale can possibly occur in patients without vascular risk factors most probably because of absence of collaterals in the brain and non response of brain haemodynamics. Smoking doubles the risk of ischaemic stroke and occurred in younger ages when compared to non smokers.

Diabetic patients with stroke were younger than non-diabetic patients with stroke[32]. Younger age in diabetic patients with stroke can be explained by diabetes mellitus accelerate atheroma formation and hyperglycemia accelerate and aggravate brain ischaemia. Transient ischaemic attacks were more frequent among diabetic patients than non-diabetic patients.

According to Amr kamel et al levels of cholesterol , triglycerides were higher in diabetic patients with stroke[32]. It is more significant for triglycerides. Dysarthria was more common in diabetic patients with stroke. Lacunar stroke syndromes were more common in diabetic stroke patients than non-diabetic stroke patients. Diabetic stroke patients had significantly worse Canadian stroke scale and non significantly worse Barthel Activity of Daily Living than non-diabetic stroke patients. In CT-Brain sub cortical infarcts are more common than cortical infarct in stroke patients with diabetics. Regarding the sex distribution, there was no difference between the diabetic stroke patients and non diabetic stroke patients. This is because diabetes mellitus has same imact on cerebral blood vessels in both sexes. Diabetes mellitus increases the permeability if blood vessel walls and causes microalbuminuria. Microalbuminuria is an independent risk factor for coronary artery disease, with the same mechanism diabetes mellitus may cause cerebrovascular disease.

Dyslipidemia have been associated with cerebrovascular atherosclerosis. Hypertriglyceridemia ti the most common abnormality seen in diabetic patients. According to Arboix et al found that hyperlipidemia was independently associated with ischemic stroke in patients with diabetes. Fibrinolytic activity in blood is regulated mainly by plasma plasminogen activator inhibitor. Increased levels of plasma plasmingen activator inhibitor present in patients with diabetes mellitus, coronary heart disease and cerebrovascular disease. Triglyceride levels correlate with plasma plasminogen activator inhibitor.

Poor metabolic control accelerates the microvascular disease, but its role in macrovascular complication in non insulin dependent diabetes mellitus is still controversial. Hyperglycemia associated with atherogenic lipoprotein changes and also it is a pro-coagulant state. Hyperglycemia decrease prostacyclin formation and increases the chance of thrombosis formation. Hyperglycemia cause glycosylation of proteins in vessel wall. These above mentioned derangements leads to accelerated atherosclerosis and increase the risk of stroke.

The influences of diabetes on various vascular lesions that cause brain ischemia

1. atherosclerosis of large intracranial arteries.

2. intracranial atheromatous branch disease of macroscopically visible branches of the intracranial arteries.

3. degenerative abnormalities such as lipohyalinosis and fibrinoid changes within penetrating artery branchs visble only microscopically

All these three above said can lead to subcortical infarct in brain parenchyma, the predominant infarct in patients with diabetes mellitus.

Diabetes increases the severity of stroke. Barett-connor-Khaw stated that it is possible that predisposes not to stroke per se but more irreversible brain damage during ischemia. Horowitz et al found that lesion size in CT-Brain and haemorrhagic transformation correlates with glucose level. Baird et al. stated that persistent hyperglycemia on serial glucose monitoring is an independent determinant of infarct expansion and worse clinical outcome.

MATERIALS AND METHODS

MATERIALS AND METHODS :

This study was conducted in tirunelveli medical college. Patients are selected from medical wards, after getting an oral consent , from july 2014 to july 2015.

INCLUSION CRITERIA :

All acute stroke patients admitted in medical wards in tirunelveli medical college hospital.

EXCLUSION CRITERIA :

1. Residual stroke patients.
2. Patients with other comorbidities like sepsis , trauma , acute cns infection , encephalopathy.

A detailed history regarding the onset of symptoms and duration of symptoms, associated symptoms like seizures, loss of consciousness, headache, vomiting were asked. Detailed history regarding, motor weakness, sensory disturbances, cerebellar autonomic disturbances, cranial nerve were asked. History regarding the status of diabetes, if diabetic duration if diabetes and his medication details are asked. Vital signs were recorded. Patients were examined according to national institute of health stroke scale and they were classified as minor stroke symptoms, mild stroke, moderate stroke, severe stroke. Patients were

examined and scored according to the performance of all 11 items in the NIH stroke scale.

METHODS OF SCORING NIH STROKE SCALE:

1.a. Level of consciousness : scored according to whether the patient is awake spontaneously(score-0), or require mild stimulation (score-1), deep painful stimulation (score-2), or patient responses with only reflex motor or autonomic reflex / totally unresponsive (score-3).

1.b. LOC questions : patients age not the date of birth,curret month were asked. If patient answers both questions correctly(score-0), one question correctly (score-1), answers neither questions correctly (score-2).

1.c. LOC Commands : ask the patient to open and close the eyes and grip the hand to command. If the patient does both the commands (score-0), patient able to perform one command (score-1), not able to perform either (score-2).

2. Best gaze (horizontal eye movement): assess the patient's ability to follow a finger or pen side to side. If it is normal (score-0), partial gaze palsy/ patient can gaze to side of infarct but can't go past midline (score-1), total gaze palsy/forced deviation/ when the gaze is fixed to one side that is not overcome by oculocephalic maneuver (score-2).

3. visual field : visual field is tested by confrontation method,if patient cannot speak use visual threat. If no visual loss (score-0), if partial hemianopia/quadrantanopia (score-1), if complete hemianopia (score-2), bilateral hemianopia or complete blindness in both eyes including cortical blindness(score---3).

4. Facial palsy : ask the patient to raise eyebrows and show teeth. Normal symmetrical movements in both sides (score-0), partial paralysis/ total or near total paralysis of lower half of face(score-1), complete paralysis of one or both sides/absence of facial movements in both upper and lower half of face(score-2).

5. Motor arm : ask the patient to lift the upper limb(each limb separately) with palm down at ninety degree and hold it for 10 seconds. If no drift for full 10 seconds score-0, able to lift the limb but drift occurs before 10 seconds, does not hit bed score-1, some effort against gravity/ limb drift down to bed score-2, no effort against gravity score-3, no movement at all score-4. This item tested and scored separately for both the upper limbs.

6. Motor leg : ask the patient to hold the leg at thirty degree for 5 seconds. If there is no drift score-0, drift occurs before 5 seconds and does not hit the bed score-1,leg falls down to the bed but there is effort against gravity score-2, no effort against gravity score-3,no movements

at all score-4. This item tested and scored separately for both lower limbs.

7. Motor coordination/limb ataxia : these item tested with eyes open. Test for both sides. If the limb is paralysed score-0,if patient can not understand score-0 given. If ataxia absent score-0, if ataxia present in one limb score-1,if ataxia present in both the limbs score-2.

8. Sensory : This item is scored according to the patient's ability to sense the pinprick. Test the face, arm (not hands), legs. patient in coma automatically given a score of 2. If there is no sensory loss score -0, score-1 is given if there is mild to moderate sensory loss but patient being aware of the touch. Score-2 for severe or total sensory loss/ patient being not aware of touch in face, arm or legs.

9. Best language : by these time patient's comprehensive ability can be assessed by preceding test results. This item is scored by asking the patient, what is happening in the picture that is attached with the NIHSS scoring sheet. Score 3 given automatically for coma patients. Score 0 given if there is no aphasia. Score 1 is given if there is mild to moderate aphasia. There is obvious reduction of fluency and/ or comprehension but patient able to make a conversation about provided materials. Score 2 is given if there is severe aphasia. All communication is through fragmentary expression. Examiner can not

identify materials provided by patient's response. Score 3 is given if there is global aphasia.

10. Dysarthria : This item is tested and scored by asking the patient to read or repeat the sentence from the attached sheet in NIHSS sheet. Score 0 is given for normal. Score 1 is given if there is mild to moderate dysarthria/ slurring present but can be understood with some difficulty. Score 2 is given if there is severe dysarthria/ speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric.

11. Extinction and inattention / formerly neglect : this item measures both the visual and sensory integration. Hold the fingers on both sides in the upper or lower quadrants of the patient's visual field and ask the patient which finger is wiggling right or left or both. To determine sensory perception of hemispheres- have the patient closed his eyes then touch his/her face, arm, leg alternating right/left/both sequence. Score them only if they can actually indicate which side is being touched. Score 0 is given if there is no abnormality. Score 1 is given if there is loss to either vision or sensory modality. Score 2 is given if both sensory and vision loss present/ does not recognize own hand or orients to only one side of face.

Patient scored on the day of admission and classified into (1) no stroke symptoms if score 0, (2) minor stroke 0-4, (3) moderate stroke if score 5 to 15, (4) moderate to severe stroke if score 16 to 20, (5) severe stroke if score 21 to 42.

Patient's random blood sugar on admission, fasting and postprandial blood sugar on next day taken. RFT, sodium, potassium, urine acetone done. Ecg, CT-Brain on admission taken.

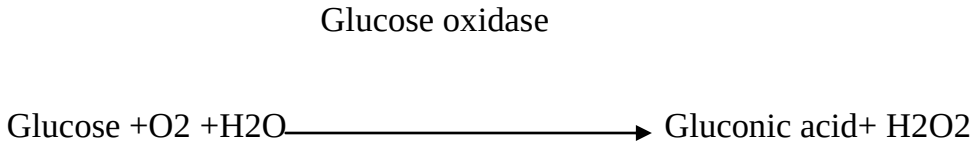
Fasting blood sugar tested after a overnight fasting of 8 hours with adequate hydration. For post prandial glucose testing blood drawn after 2 hours of food intake. Blood sugar estimated by TRINDER'S method.

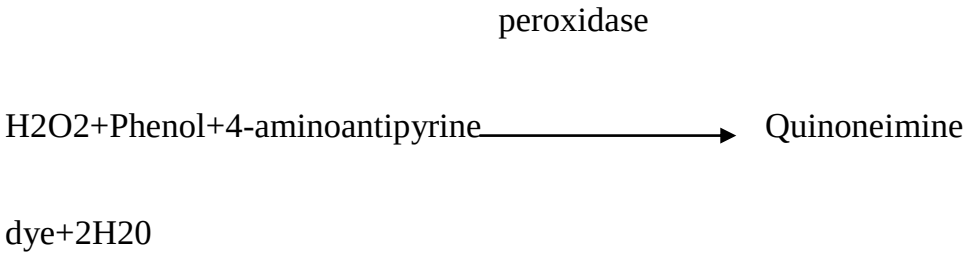
TRINDER'S METHOD :

Glucose estimation by this method is explained by Trinder in 1969. It has almost all the attributes of an ideal automated colorimetric glucose oxidase procedure.

PRINCIPLE :

In the presence of catalytic enzyme glucose oxidase, Glucose in sample is oxidized to yield gluconic acid and hydrogen peroxide. The peroxidase enzyme catalyses the oxidative coupling of 4-aminoantipyrine with Phenol to yield a colured quinoneimine complex. Quinoneimine complex absorbance proportional to the concentration of glucose in sample.





REAGENT COMPOSITION

REAGENT 1: ENZYME REAGENT

ACTIVE INGREDIENT	CONCENTRATION
Glucose oxidase	>/=20000 U/L
Peroxidase	>/= 2000 U/L
Phenol	10 mmol/L
Phosphate buffer	200mmol/L

Glucose standard 100mg/dl.

Specimen : to prevent the glycolysis serum to be separated as soon as possible.

sodium fluoride to be added to the sample to inhibit glycolysis.

ASSAY PROCEDURE (end point) :

Pipette into test tube labeled as	Blank	Standard	Test
sample	----	-----	10 microL
standard	----	10 microL	-----
Enzyme reagent	1.0 ml	1.0 ml	1.0 ml

CALCULATION :

Glucose= $absorbance\ of\ test/absorbance\ of\ standard \times concentration\ of\ standard \quad (mg/dl)$

Normal values reference.

Fasting 65-110 mg/dl ; Postprandial 90-130 mg/dl.

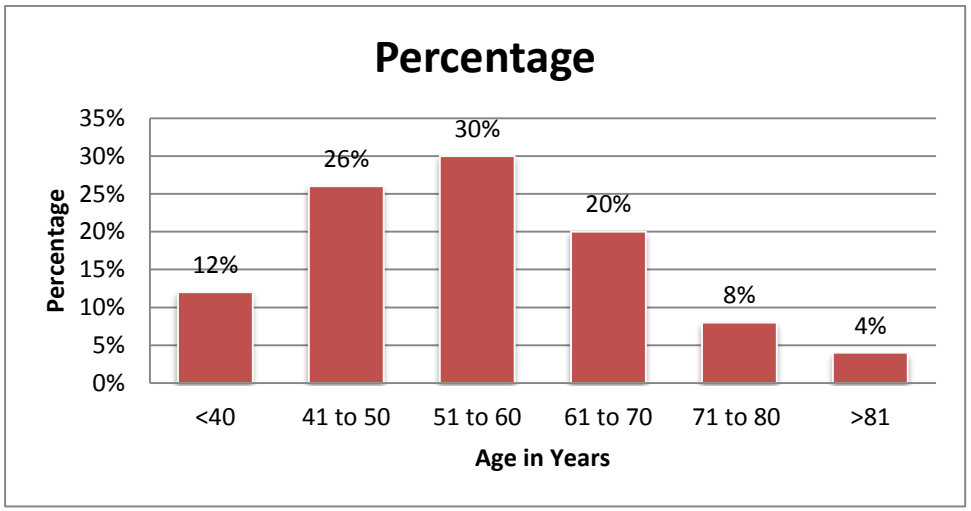
Observation and Results

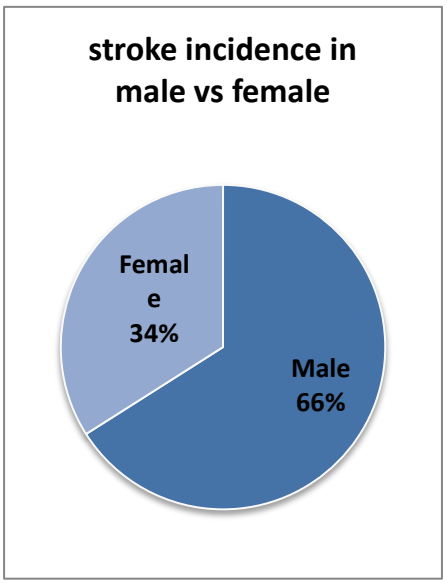
OBSERVATION AND RESULTS :

In this study stroke incidence regarding age given in the below chart. Maximum incidence in 51-60 years of age (30% of study population). Followed by 41-50 years (26% 0f study population), then 61-70 years of age (20% of study population).

Age	Patients	Percentage
<40	6	12%
41-50	13	26%
51-60	15	30%
61-70	10	20%
71-80	4	8%
>81	2	4%

STROKE INCIDENCE AND ITS AGE DISTRIBUTION

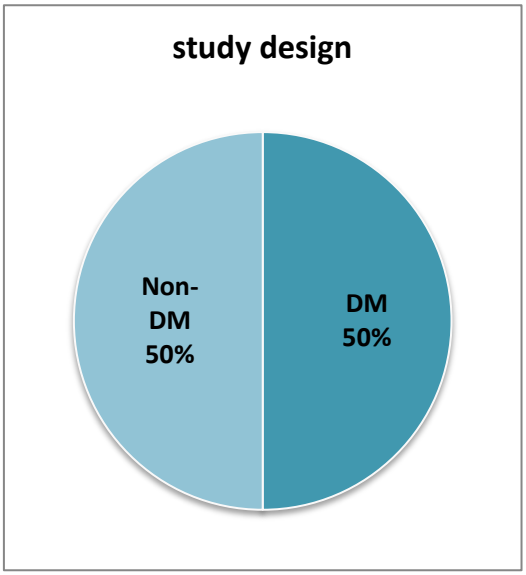




stroke incidence in relation to sex in this study .

Male	33
female	17

STUDY DESIGN



DM	25
NON - DM	25

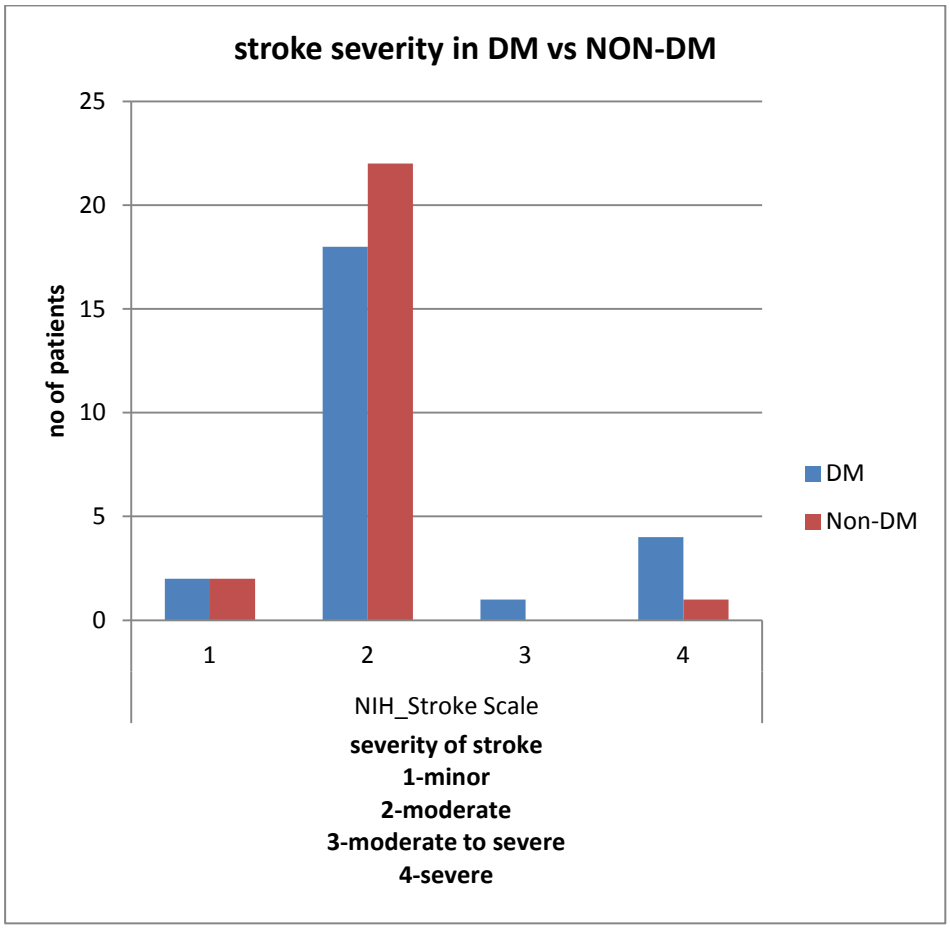
Study design : stroke in 50% of DM, stroke in 50% of NON-DM were taken in this study.severity of stroke in each of this population analysed. Stroke severity in impaired glucose tolerance patients analysed.

STROKE SEVERITY IN DIABTIC vs NON-DIABETIC PATIENTS :

Among 25 diabetic patients Minor stroke occurred in 2 patients , moderate stroke occurred in 18 patients , moderate to severe stroke occurred in 1 patient , severe stroke occurred in 4 patients. Among non-diabetic patients minor stroke occurred in 2 patients , moderate to severe stroke occurred in 22 patients , severe stroke occurred in 1 patient. Stroke severity measured by NIH stroke scale.

STROKE SEVERITY IN DM vs NON-DM PATIENTS BY NIH STROKE SCALE :

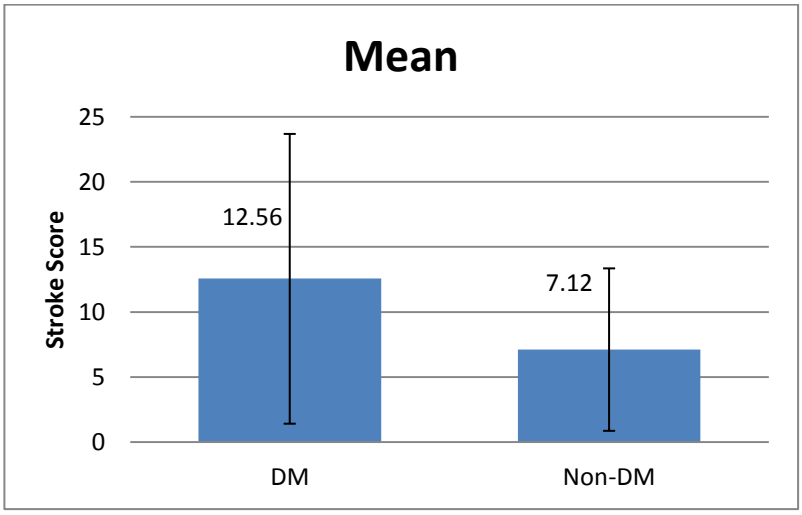
	NIH_Stroke Scale			
	1	**2**	**3**	**4**
DM	2	18	1	4
Non-DM	2	22	0	1



StrokeTotalScorebyNIHSS

	Mean	S.D	P value
DM	12.56	11.13	
Non-DM	7.12	6.24	**0.038**

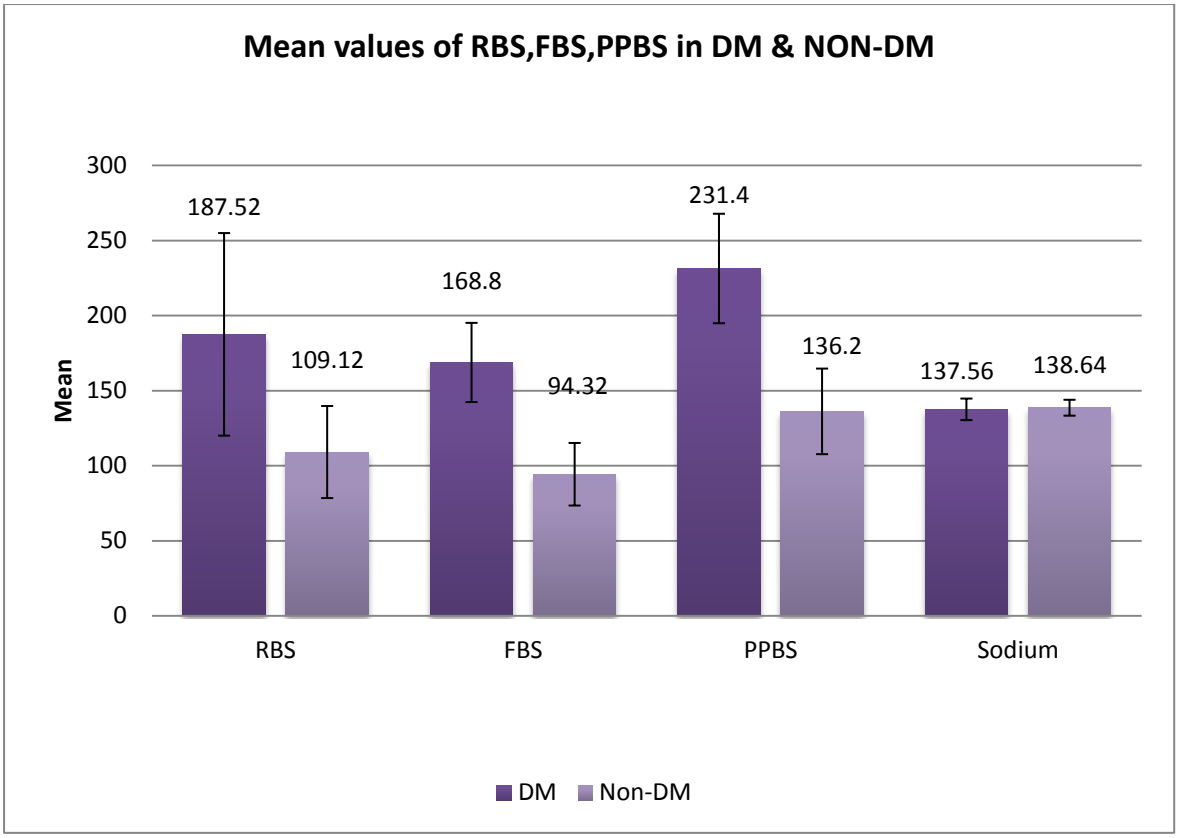
Mean score of NIH stroke scale in diabetic patients is 12.56 and standard deviation is 11.13. Mean score of NIH stroke scale in non-diabetic patients is 7.12 and standard deviation is 6.24 . By independent sample t test p value P < 0.038 . there is statistically significant difference in stroke severity between diabetic stroke patients and non-diabetic patients .



COMPARING NIHSS AND THEIR BLOOD SUGAR VALUES

	DM		Non-DM		
	Mean	**S.D**	**Mean**	**S.D**	**P value**
RBS	187.52	67.47	109.12	30.67	<0.0001
FBS	168.8	26.37	94.32	20.84	<0.0001
PPBS	231.4	36.47	136.2	28.51	<0.0001
Sodium	137.56	7.17	138.64	5.3	0.548
NIH_Score	12.56	11.13	7.12	6.24	0.038

Independent sample t Test.



The above chart shows the mean values of RBS , FBS , PPBS levels in Diabetic and non-diabetic stroke patients. There is statistically significant correlation between severity of stroke in diabetic patients and their Random blood sugar and fasting blood sugar .

PEARSON CORRELATION TEST :

	Stroke Score			
	DM		Non-DM	
	Coefficient correlation	p value	Coefficient correlation	p value
RBS	0.592	0.002	0.169	0.419
FBS	0.435	0.03	-0.05	0.813
PPBS	0.27	0.192	-0.071	0.736
Sodium	-0.808	<0.0001	-0.588	0.002

By Pearson correlation test there is statistically significant correlation between random blood sugar severity of stroke in diabetic patients. There is a linear relationship between random blood sugar and severity of stroke. Also there is statistically significant correlation between fasting blood sugar and severity of stroke in diabetic patients. There is linear relationship between FBS and severity of stroke in diabetic patients. No statistically significant correlation between postprandial blood sugar and stroke severity.

STROKE SEVERITY IN IMPAIRED GLUCOSE TOLERANCE :

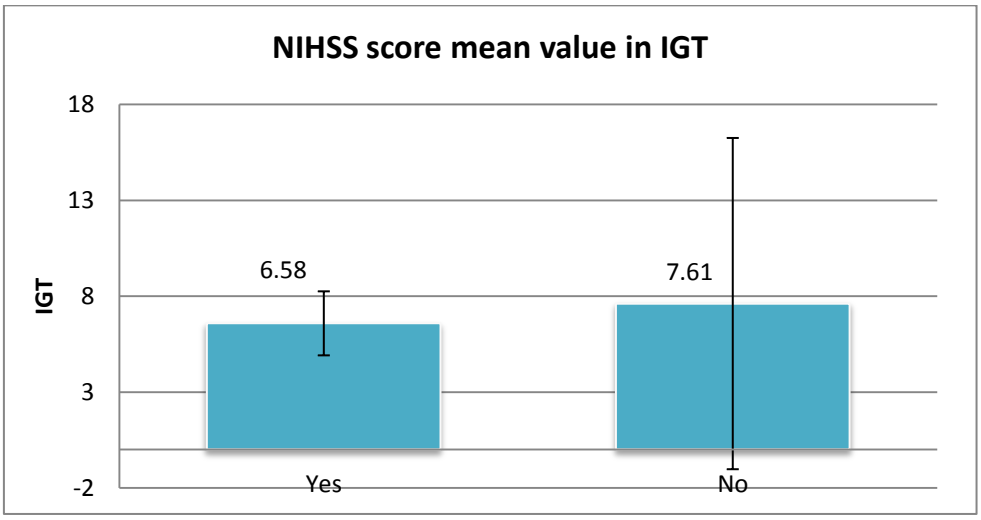
		NIH_Stroke Scale			
		1	**2**	**3**	**4**
IGT	**Yes**	0	12	0	0
	No	2	10	0	1

Stroke patients with impaired glucose tolerance in this study had moderate stroke severity according to NIHSS in this study.

IGT

IGT	**Mean**	**S.D**
Yes	6.58	1.67
No	7.61	8.64

In this study twelve patients had impaired glucose tolerance. The above table shows the mean value of the NIHSS severity score. There is no statistically significant difference between the IGT stroke patients and stroke patients with euglycemia.



The above chart shows the mean value of NIH stroke scale in impaired glucose tolerance patients. Mean NIHSS in IGT is 6.58 ,Mean NIHSS in euglycemic is 7.61. No statistically significant increase in severity of stroke in Impaired glucose tolerance patients.

INCIDENCE OF SEVERE STROKE IN DM VS NON-DM PATIENTS :

	Severe Stroke	
	Yes	No
DM	4	21
Non-DM	1	24

In this study five patients had severe stroke according to NIHSS(NIHSS > 21). Among them four patients are diabetic ,one patient is non-diabetic. So there is evidence of severe stroke incidence in diabetics are more than non-diabetic patients.

SEVERE STROKE AND HYPONATREMIA :

		Severe Stroke			
		Yes	**No**	**P value**	Fisher's exact test
Sodium <130	**Yes**	4	1	<0.0001	
	No	1	4		

Among five severe stroke patients, four patients had decreased sodium. There is statistically significant stroke severity in hyponatremia.

DISCUSSION

DISCUSSION :

Stroke is the important cause of mortality and morbidity in worldwide. Risk factors for stroke are increased age , male , systemic hypertension, Diabetes mellitus, Dyslipidemia , Renal and cardiac disease , smoking , alcoholics , obesity , physical inactivity. Most of the existing studies regarding severity of stroke in diabetics were conducted in developed countries and large cities of developing countries like india. This study has been conducted predominantly in rural population in and around Tirunelveli.

In our study stroke incidence is more common in male patients than female patients. Incidence of stroke in male patiets are 66% and incidence of stroke in female patients are 34%. Most common age groups are 51-60 years (30%) , 41-50 years (26%), 61-70 years (20%). According to R P Eaten et al , the stroke incidence was more common in males. Most common age groups are 51-60 rears of age.

In our study the severity of stroke is more in diabetic patients . There is linear relationship between Random blood sugar on admission and severity of stroke . similar results found in Ahmed Hussein et al ,hyperglycemia level at the time of admission influences the severity of stroke[33]. In our study high Random blood sugar level taken at the time

of admission high NIH stroke scale score found in diabetic stroke patints ($P < 0.02$). Also there is linear relationship between fasting bloodsugar and NIH stroke scale score ($P < 0.03$). According to Clara Hjalmarsson et al , poor glycemic control prior to ischaemic stroke is an independent risk factor for poor survival ,and a marker for stroke severity (high NIHSS on admission) and associated with poor outcome[34].

According to Copenhagen stroke study , Henrik stig jogensen et al , diabetic stroke patients are younger than non-diabetic stroke patients[35]. They had hypertension more frequently. Occurrence of intracerebral haemorrhage as the cause of stroke in diabetic patients is less common . In our study among 50 patients , four patients had intracerebral haemorrhage. Among 25 diabetic patients only 2 patients had intracerebral haemorrhage , 23 patients had ischaemic stroke. This study also shows the incidence of intracerebral haemorrhage as the cause of stroke is less common.

K Ghanachandra Singh et al , the incidence of stroke is more common in the age group of 51-60 years which is in agreement with our study[36]. Higher mortality with stress hyperglycemics and diabetics. Recovery of ATP generation impaired in stroke patients with hyperglycemia is impaired. It was stated there is increased adhesiveness of red blood cells in diabetic stroke patients.

According to Karl Matz et al , patients with diabetes had more severe stroke attacks when compared to non-diabetic stroke patients , impaired glucose tolerance patients and transient hyperglycemic patients[30]. Higher rate of infectious complications and worse outcome in diabetic stroke patients. In our study also shows occurrence of more severe stroke in diabetic patients. But in our study there is no statistically significant severity of stroke in impaired glucose tolerance.

According to Amr Kamel et al , diabetic patients were younger than non-diabetic stroke patients[32]. Stroke patients with diabetes had worse Canadian stroke scale. In our study stroke patients with diabetics had severe/worse NIH stroke scale. According to Amr Kamel et al diabetic stroke patients had high number of lacunar stroke.

According to the study by Sheikh saleem et al, out of 1000 stroke patients 353 had hyponatremia. Their mortality rate when compared to patients with normal sodium level were high and statistically significant. In our study among five severe stroke patients 4 patints had hyponatremia. it is in agreement with the above study that hyponatremia influences the severity of stroke.

SUMMARY

AND

CONCLUSION

SUMMARY & CONCLUSION :

In our study it was found ,

- Males were more commonly affected than females.
- Age group 51-60 years more commonly affected.
- Diabetic patients had more severe stroke than non-diabetic patients.
- Random blood sugar on admission had linear relationship with NIH stroke scale in diabetic patients.
- Fasting blood sugar also had linear relationship with NIH stroke scale in diabetic patients.

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ANNEXURES

Annexure 1

Clinical proforma

Personal details

NAME : AGE: SEX:

OCCUPATION:

ADDRESS:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST HISTORY:

FAMILY HISTORY:

GENERAL EXAMINATION:

VITAL SIGNS:

CLINICAL EXAMINATION:

NIH STROKE SCALE

NIH STROKE SCALE

Patient Identification. ___ ___-___ ___ ___-___ ___ ___

Pt. Date of Birth ___ ___/___ ___/___ ___

Hospital _____(___ ___-___ ___)

Date of Exam ___ ___/___ ___/___ ___

Person Administering Scale _____

Administer stroke scale items in the order listed. Record performance in each category after each subscale exam. Do not go back and change scores. Follow directions provided for each exam technique. Scores should reflect what the patient does, not what the clinician thinks the patient can do. The clinician should record answers while administering the exam and work quickly. Except where indicated, the patient should not be coached (i.e., repeated requests to patient to make a special effort).

1a. Level of Consciousness: The investigator must choose a response if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, orotracheal trauma/bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious	0 = **Alert;** keenly responsive. 1 = **Not alert**; but arousable by minor stimulation to obey, answer, or respond. 2 = **Not alert**; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not	
1b. LOC Questions: The patient is asked the month and his/her age. The answer must be correct - there is no partial credit for being close. Aphasic and stuporous patients who do not comprehend the questions will score 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner not "help" the patient with verbal or non-verbal cues.	0 = **Answers** both questions correctly. 1 = **Answers** one question correctly. 2 = **Answers** neither question correctly.	
1c. LOC Commands: The patient is asked to open and close the eyes and then to grip and release the non-paretic hand. Substitute another one step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime), and the result scored (i.e., follows none, one or two commands). Patients with trauma, amputation, or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.	0 = **Performs** both tasks correctly. 1 = **Performs** one task correctly. 2 = **Performs** neither task correctly.	

NIH STROKE SCALE

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2. Best Gaze: Only horizontal eye movements will be tested. Voluntary or reflexive (oculocephalic) eye movements will be scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score will be 1. If a patient has an isolated peripheral nerve paresis (CN III, IV or VI), score a 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, pre-existing blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.	0 = **Normal.** 1 = **Partial gaze palsy;** gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present. 2 = **Forced deviation,** or total gaze paresis not overcome by the oculocephalic maneuver.	

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3. Visual: Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If patient is blind from any cause, score 3. Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a 1, and the results are used to respond to item 11.	0 = **No visual loss.** 1 = **Partial hemianopia.** 2 = **Complete hemianopia.** 3 = **Bilateral hemianopia** (blind including cortical blindness).	
4. Facial Palsy: Ask – or use pantomime to encourage – the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or non-comprehending patient. If facial trauma/bandages, orotracheal tube, tape or other physical barriers obscure the face, these should be removed to the extent possible.	0 = **Normal** symmetrical movements. 1 = **Minor paralysis** (flattened nasolabial fold, asymmetry on smiling). 2 = **Partial paralysis** (total or near-total paralysis of lowerface). 3 = **Complete paralysis**	
5. Motor Arm: The limb is placed in the appropriate position: extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Drift is scored if the arm falls before 10 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic arm. Only in the case of amputation or joint fusion at the shoulder, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.	0 = **No drift;** limb holds 90 (or 45) degrees for full 10 seconds. 1 = **Drift;** limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. 2 = **Some effort against gravity;** limb cannot get to or maintain (if cued) 90 (or 45) degrees, drifts down to bed, but has some effort against gravity. 3 = **No effort against gravity;** limb falls. 4 = **No movement.** UN = **Amputation** or joint fusion, **5a. Left Arm** **5b. Right Arm**	
6. Motor Leg: The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Drift is scored if the leg falls before 5 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic leg. Only in the case of amputation or joint fusion at the hip, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.	0 = **No drift;** leg holds 30-degree position for full 5 seconds. 1 = **Drift;** leg falls by the end of the 5-second period but does not hit bed. 2 = **Some effort against gravity;** leg falls to bed by 5seconds, but has some effort agains tgravity. 3 = **No effort against gravity;** leg falls to bed immediately 4 = **No movement .**UN = **Amputation** or joint fusion **6a. Left Leg**	

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7. Limb Ataxia: This item is aimed at finding evidence of a unilateral cerebellar lesion. Test with eyes open. In case of visual defect, ensure testing is done in intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice. In case of blindness, test by having the patient touch nose from extended arm position.	0 = **Absent.** 1 = **Present in one limb.** 2 = **Present in two limbs.** UN = **Amputation** or joint fusion,	
8. Sensory: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal and the examiner should test as many body areas (arms [not hands], legs, trunk, face) as needed to accurately check for hemisensory loss. A score of 2, "severe or total sensory loss," should only be given when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will, therefore, probably score 1 or 0. The patient with brainstem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score2. Patients in a coma (item 1a=3) are automatically given a 2 on thisitem.	0 = **Normal;** no sensory loss. 1 = **Mild-to-moderate sensory loss;** patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched. 2 = **Severe to total sensory loss;** patient is not aware of being touched in the face, arm, and leg.	
9. Best Language: A great deal of information about comprehension will be obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet and to read from the attached list of sentences. Comprehension is judged from responses here, as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a=3) will automatically score 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of3 should be used only if the patient is mute and follows no one-step commands.	0 = **No aphasia;** normal. 1 = **Mild-to-moderate aphasia;** some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient's response .2 = **Severe aphasia;** all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response. 3 = **Mute, global aphasia;** no usable speech or auditory comprehension.	
10. Dysarthria: If patient is thought to be normal, an adequate sample of speech must be obtained by asking patient to read or repeat words from the attached list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech, the examiner should record the score as untestable (UN), and clearly write an explanation for this choice. Do not tell the patient why he or she is being tested.	0 = **Normal.** 1 = **Mild-to-moderate dysarthria;** patient slurs at least some words and, at worst, can be understood with some difficult .2 = **Severe dysarthria;** patient's speech is so slurred as to beunintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric .UN = **Intubated** or other physical barrier,	

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11. Extinction and Inattention (formerly Neglect): Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe visual loss preventing visual double simultaneous stimulation, and the cutaneous stimuli are normal, the score is normal. If the patient has aphasia but does appear to attend to both sides, the score is normal. The presence of visual spatial neglect or anosagnosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never untestable.	0 = No abnormality 1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities. 2 = Profound hemi-inattention or extinction to more than one modality; does not recognize own hand or orients to only one side of space.	

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s.no	ip no	name	age	sex	DM-1,NON DM-2	impaired glucose tolerance IGT-1,non IGT-2	Random blood sugar	urea	creatinine	sodium	potassium	fbs	ppbs	urine acetone	hb(gm%)	platelet	ecg	ct brain	nihss	level of consciouness	LOC QUESTIONS	LOC COMMANDS	BEST GAZE	VISUAL	FACIAL PALSY	MOTOR ARM-L	MOTOR ARM-R	MOTOR LEG-L	MOTOR LEG R	LIMB ATAXIA	SENSORY	BEST LANGUAGE	DYSARTHRIA	EXTINCTION&INATTENTION		total score
1	**46740**	Alaganantham	52	M	1		159	26	0.6	140	4.2	**149**	**364**	negative	14.6	2.72	normal	rt capsuloganglionic infarct		1	0	0	0	0	2	**3**	0	**3**	0	0	0	0	0	0		9
2	46778	Murugaiah	60	M	2	1	82	34	0.4	141	3.8	68	186	negative	16	1.39	coronary artery disease	Lt fronto parietal region haemorrhage		2	1	1	0	0	2	0	2	0	2	0	0	1	0	0		11
3	**26852**	LAKSHMANAN	67	M	1		**204**	39	1.2	138	4.1	**165**	**234**	negative	14	1.98	normal	Lt capsulo ganglionic infarct		1	1	1	0	0	2	0	3	0	3	0	0	1	0	0		12
4	45235	Ponnusamy	52	M	2	1	125	47	1	140	4.4	102	147	negative	16.1	2.36	normal	rt lateral medullary infarct		0	0	0	0	0	0	0	0	0	0	2	2	0	2	0		6
5	**23810**	Ammasi	42	M	1		**241**	39	1.3	124	4.2	**181**	**284**	negative	13.2	1.75	normal	Lt MCA territory infarct		3	2	2	1	3	3	4	4	4	4	0	2	3	2	2		39
6	42515	Rasathi	45	F	2	1	219	36	1.2	134	4.2	117	146	negative	11.5	2.34	atrial fibrillation	rt capsuloganglionic infarct		0	0	0	0	0	2	3	0	3	0	0	0	0	0	0		8
7	43921	Kulanthaivelu	48	m	2	2	130	18	0.7	124	4	96	106	negative	10.6	1.75	normal	Lt MCA territory infarct		2	2	2	1	3	2	4	4	4	4	0	1	3	2	2		36
8	**42270**	Salim	55	M	1		**425**	33	2	123	4.5	**245**	**273**	negative	13.5	3.8	Lt ventricular hypertrophy,cad	multi infarct		2	2	2	1	3	2	4	4	4	4	0	1	3	2	2		36
9	**42821**	Paneer pandi	48	M	1		107	22	0.6	121	3.4	**215**	**242**	negative	14.6	2.73	CAD	intracerebral haemorrhage -Lt		2	2	2	1	3	2	4	4	4	4	0	1	3	2	2		36
10	46775	ESAKKIAMMAL	77	f	2	2	89	20	0.9	147	3.8	105	114	negative	13.3	2.63	CAD	B/L thalamic infarct		1	1	0	0	0	0	0	0	0	0	0	1	1	1	1		6
11	**46821**	kuthalathammal	55	F	1		348	141	3.9	133	5.9	**141**	**203**	negative	13.1	5.05	LVH	Lt-capsulo ganglionic haemorrhage		2	2	2	1	3	2	4	4	4	4	0	1	3	2	2		36
12	48229	Rajan	68	M	2	2	121	66	1.2	150	4.4	123	104	negative	14.6	2.47	normal	Lt-thalamic bleed		0	0	0	0	0	2	1	0	1	0	0	1	0	0	0		5
13	40826	balakrishnan	46	M	2	2	107	23	0.9	139	4.2	112	75	negative	13.2	2.17	normal	infarct Lt-corona radiata		0	0	0	0	0	0	1	0	1	0	0	0	0	0	0		2
14	40824	karuppasamy	62	M	2	2	124	29	1.2	139	4.2	102	101	negative	14.1	1.98	incomplete RBBB	infarct Rt-capsuloganglionic region		0	0	0	0	0	2	1	0	1	0	0	0	0	0	0		4
15	**37951**	Muthulakshmi	68	F	1		181	51	1.2	146	4.6	**161**	**242**	negative	12.4	2.14	normal	Lt-capsulo ganglionic infarct		1	1	1	0	0	2	0	3	0	3	0	0	1	0	0		12
16	37981	vinayagathammal	79	F	2	2	121	18	1.9	139	4.1	82	138	negative	13.3	2.86	normal	posterior circulation stroke		0	0	0	1	1	0	1	1	1	1	1	0	0	0	1		8
17	**41019**	kulathuran	58	m	1		139	224	10.1	136	5.3	**161**	**201**	negative	9.7	1.74	LVH	Lt-capsulo ganglionic infarct		0	0	0	0	0	2	0	2	0	2	0	0	1	0	0		7
18	**40827**	Ramzan kani	35	M	1		157	18	0.7	141	4	**121**	**202**	negative	12	1.81	normal	Rt cerebellar infarct		0	0	0	0	0	0	0	0	0	0	2	0	0	1	0		3
19	**40868**	mariappan	40	M	1		143	40	1.4	139	4.2	**162**	**204**	negative	13.4	2.13	normal	Lt capsulo ganglionic infarct		0	0	0	0	0	2	0	2	0	2	0	0	1	0	0		7
20	43928	kumar	42	M	2	1	97	56	0.9	142	4	60	146	negative	17.4	3.2	normal	rt capsuloganglionic infarct		0	0	0	0	0	2	2	0	2	0	0	0	0	0	0		6
21	46839	subramaniyan	60	m	2	1	83	16	1.2	137	4	74	150	negative	17.1	0.88	normal	Lt fronto temporal infarct		0	0	0	0	0	1	0	2	0	2	0	0	0	0	0		5
22	**43925**	syed ali fathima	87	F	1		151	59	2.4	127	4.1	**142**	**204**	negative	6	0.65	LVH	Lt fronto temporal infarct		2	2	2	0	1	1	0	3	0	3	0	0	1	1	1		17
23	39388	muthuvel	55	M	2	2	62	21	1	135	4.1	64	86	negative	12	1.83	normal	lt.capsulogan glionic infarct		0	0	0	0	0	1	0	2	0	2	0	0	0	0	0		5
24	**39359**	panakottai pandiyan	75	M	1		112	62	2.2	138	4.2	**145**	**207**	negative	14.2	2.13	LVH	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	3	0	3	0	0	0	0	0		7

25	**20788**	thangappan	62	M	1		213	37	1.1	141	4.3	**162**	**217**	negative	13.7	2.1	normal	rt capsulogangli onic infarct		0	0	0	0	0	1	3	0	3	0	0	0	0	0	0		7
26	**25319**	somasundaram	48	M	1		201	44	1.4	145	4.6	**171**	**234**	negative	12.9	2.3	LVH	lt.capsulogan glionic infarct		1	0	0	0	0	1	0	3	0	3	0	0	0	0	0		8
27	**29983**	rajendran	56	M	1		167	37	0.9	138	3.9	**201**	**261**	negative	13	2.43	LVH	lt.capsulogan glionic infarct		1	0	0	0	0	1	0	3	0	3	0	0	1	0	0		9
28	**47751**	kasi	52	M	1		181	41	1.3	141	4.2	**212**	**248**	negative	12.7	2.11	normal	lt.capsulogan glionic infarct		1	0	0	0	0	1	0	2	0	3	0	0	1	0	0		8
29	**31860**	arumugam	41	M	1		167	39	1.1	131	3.6	**167**	**204**	negative	10.8	1.98	normal	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	3	0	3	0	0	0	0	0		7
30	**27923**	abubakar siddique	37	M	1		182	31	0.6	141	3.9	**159**	**212**	negative	11	2.14	normal	Lt capsulo ganglionic infarct		1	0	0	0	0	1	0	3	0	3	0	0	1	0	0		9
31	**28980**	subburaj	33	M	1		163	39	0.9	139	3.9	**161**	**212**	negative	13	2.32	normal	rt capsulogangli onic infarct		0	0	0	0	0	1	2	0	2	0	0	0	0	0	0		5
32	**33299**	perumal	60	F	1		159	36	1.9	142	4.3	**162**	**206**	negative	12.7	2.21	LVH	Lt capsulo ganglionic infarct		1	0	0	0	0	1	0	3	0	3	0	0	0	0	0		8
33	**30057**	krishnan	70	M	1		197	46	1.4	145	3.8	**181**	**233**	negative	13.2	2.32	normal	Lt capsulo ganglionic infarct		1	0	0	0	0	1	0	2	0	3	0	0	0	0	0		7
34	**10638**	murugan	43	m	1		163	38	1.2	138	3.1	**151**	**201**	negative	14.1	2.13lakh	normal	Lt capsulo ganglionic infarct		1	0	0	0	0	1	0	3	0	3	0	0	0	0	0		8
35	**12049**	samuthiram	63	M	1		171	41	1.3	145	4.6	**162**	**216**	negative	12.8	1.99	normal	Rt cerebellar infarct		0	0	0	0	0	0	0	0	0	0	2	0	0	1	0		3
36	**9041**	kadar sherif	50	M	1		183	43	1.5	144	4.4	**172**	**247**	negative	11.6	2.43	normal	Rt capsulo ganglionic infarct		0	0	0	0	0	1	3	0	3	0	0	0	0	0	0		7
37	**10501**	paramasivan	55	M	1		174	36	1.2	143	4.1	**171**	**234**	negative	12.5	1.99	normal	Rt capsulo ganglionic infarct		0	0	0	0	0	1	3	0	3	0	0	0	0	0	0		7
38	36402	velasari	70	M	2	1	164	42	1	141	4	**122**	**160**	negative	13	2.31	normal	rt capsulogangli onic infarct		0	0	0	0	0	1	2	0	2	0	0	0	0	0	0		5
39	33278	selvaraj	54	M	2	2	98	37	1.3	138	3.8	86	139	negative	12.2	2.1	normal	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	2	0	2	0	0	0	0	0		5
40	25941	ganapathi	70	M	2	1	103	49	2.1	131	4.7	89	149	negative	10.2	1.89	LVH	Rt capsulo ganglionic infarct		0	0	0	0	0	1	3	0	3	0	0	0	0	0	0		7
41	27901	paulraj	72	M	2	2	121	39	1.5	134	3.4	99	134	negative	11.2	2.11	normal	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	3	0	3	0	0	0	0	0		7
42	27495	kalimuthu	66	M	2	2	108	29	0.9	141	3.8	94	129	negative	13.4	2.34	normal	lt.capsulogan glionic infarct		0	0	0	0	0	1	0	2	0	2	0	0	0	0	0		5
43	30256	chelladurai	45	m	2	1	112	34	0.9	139	3.8	91	143	negative	14.2	2.43	normal	Lt capsulo ganglionic infarct		1	0	0	0	0	1	0	2	0	2	0	0	1	0	0		7
44	27913	gunasekar	50	M	2	2	104	28	0.8	141	4.1	89	134	negative	13.1	2.09	normal	Rt capsulo ganglionic infarct		0	0	0	0	0	1	3	0	2	0	0	0	0	0	0		6
45	46549	jeyapal	40	M	2	2	100	31	0.9	143	4.1	78	139	negative	12.4	1.98	normal	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	2	0	2	0	0	0	0	0		5
46	33372	subbiah	85	M	2	1	101	56	2.1	130	5	87	141	negative	10.1	1.76	LVH	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	3	0	2	0	0	0	0	0		6
47	20759	perumal	56	M	2	1	89	43	1.3	140	4.1	91	152	negative	14.6	2.43	normal	Rt capsulo ganglionic infarct		0	0	0	0	0	1	2	0	2	0	0	0	0	0	0		5
48	20736	sundara pandi	60	M	2	1	92	39	1.1	142	3.9	101	156	negative	13.2	2.4	normal	Rt capsulo ganglionic infarct		0	0	0	0	0	1	2	0	3	0	0	0	0	0	0		6
49	43789	jesuraj	40	M	2	2	84	31	0.8	140	3.8	87	131	negative	13.9	2.11	normal	rt capsulo ganglionic infarct		0	0	0	0	0	1	2	0	2	0	0	0	0	0	0		5
50	43752	murugan	49	M	2	1	92	39	0.9	139	4.1	99	149	negative	13.2	2.03	normal	Lt capsulo ganglionic infarct		0	0	0	0	0	1	0	3	0	3	0	0	0	0	0		7